THE PLANT DISEASE REPORTER

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Division of Mycology and Disease Survey

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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THE PLANT DISEASE SURVEY
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EFFECTS OF SOME ANTIBIOTICS AND BACTERICIDES ON GROWTH OF XANTHOMONAS JUGLANDIS AND X. CORYLINA[1]

T. R. Aspitarte[2], W. B. Bollen[3], and P. W. Miller[4]

Walnut bacteriosis and filbert bacteriosis, caused by Xanthomonas juglandis (Pierce) Dowson and Xanthomonas corylina (Miller et al.)Starr & Burkh., respectively, are of major economic importance in the Pacific Northwest. While practical control methods have been developed for both these diseases (5, 6), they consist primarily of protective spraying, which is costly and time consuming. Methods of control that are simpler to employ, longer lasting, and less costly would be desirable.

The possibility of curing certain plant diseases by the application of chemotherapeutic agents which exert their action inside the host plants has been given increased attention in the last decade by many investigators. Considerable progress has already been made in finding materials and methods of antidoting toxins produced by certain pathogens within the host (2, 10, 11). Also, certain inhibitive agents, or antibiotics, which inactivate or prevent the multiplication of certain parasites within the host (3, 7, 9) have recently been discovered. Rudolph (8), in 1946 attempted to control X. juglandis by the injection of penicillin into infected trees but without success. Ark (1), in 1949, reported relatively poor control of walnut bacteriosis from spraying trees with streptomycin (1 p.p.m.). No trials have been carried on for the control of filbert bacteriosis by chemotherapeutic methods. Since the investigations of Rudolph (8) and Ark (1), new antibiotics which seemed worthy of trial against X. juglandis and X. corylina have been developed. There follows a discussion of plate tests in which some of the newer antibiotics and certain bactericides were tested against these two organisms.

Materials and Methods

Sterile nutrient agar plates were "seeded", respectively, with freshly made isolates of the two pathogens, the isolation technique used being that described by McNew (4). Standard discs of certain antibiotics and solutions of certain bactericides of various concentrations (Table 1) in sterile cups were placed on the surface of "seeded" plates. The plates were then incubated at 37° C. for 36 hours, the time required for untreated "seeded" plates to show the maximum amount of growth. Inhibition, as measured by cleared-zone diameters and recorded in five degrees of intensity, is shown in Table 1.

Results

As shown in Table 1, only mercuric chloride 1:500 and oxyquinoline benzoate 1:1000 completely inhibited both organisms. Aureomycin, streptomycin, terramycin, crystal violet, and Puratized Spray caused various degrees of inhibition (Figures A and B). Little or no inhibition occurred with penicillin, bacitracin, chloromycetin, sulfanilamide, and Actidione.

The two organisms studied, which are practically identical culturally, showed differences in their reaction to some of the materials. Penicillin, neomycin, polymyxin B, and chloromycetin at the higher concentrations had some inhibiting effect on X. corylina but not on X. juglandis (Table 1).

There was some indication of the development of resistant types of both organisms or else a loss of potency in the antibiotics. This was indicated by the delayed (10 to 20 days) appearance of bacterial colonies in the originally completely cleared zones around discs holding certain antibiotics (Figure C).

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[1]Approved for publication as Technical Paper No. 774 by the Director of the Oregon Agricultural Experiment Station and by the Chief of the Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Department of Agriculture. Contribution of the Departments of Bacteriology and Botany and Plant Pathology of the Oregon Agricultural Experiment Station and of Division of Fruit and Nut Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering.

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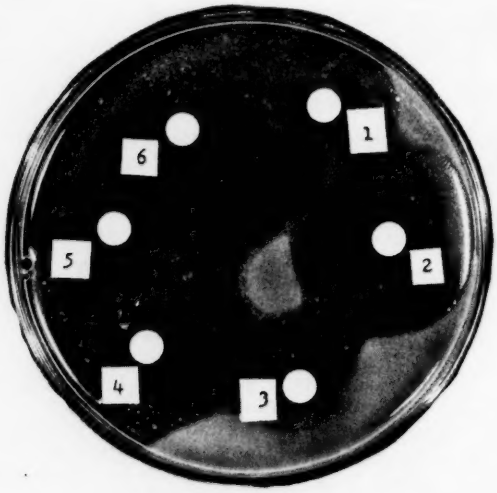

FIGURE A. Growth of Xanthomonas juglandis, 5 days after "seeding", on pure nutrient agar on which were scattered discs treated with antibiotics as follows: 1, streptomycin (10 µ g.), 2, streptomycin (100 µ g.), 3, aureomycin (2 µ g.), 4, aureomycin (20 µ g.), 5, terramycin (10 µ g.), and 6, terramycin (100 µ g.).

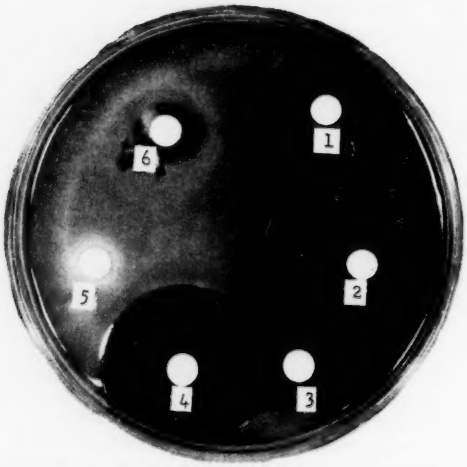

FIGURE B. Growth of Xanthomonas corylina, 5 days after "seeding", on pure nutrient agar on which were scattered discs treated with antibiotics as follows: 1, terramycin (10 µ g.), 2, terramycin (100 µ g.), 3, aureomycin (2 µ g.), 4, aureomycin (20 µ g.), 5, chloromycetin (2 µ g.), and 6, chloromycetin (20 µ g.).

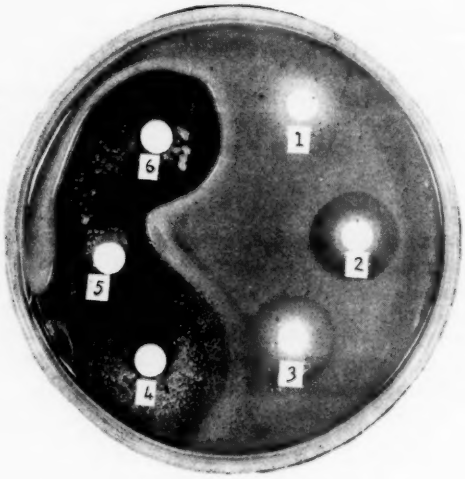

FIGURE C. Growth of Xanthomonas corylina, 12 days after "seeding", on pure nutrient agar on which were scattered discs treated with antibiotics as follows: 1, penicillin (1 unit), 2, penicillin (10 units), 2, bacitracin (2 units), 4, bacitracin (20 units), 5, streptomycin (10 µ g.), and 6, streptomycin (100 µ g.).

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Table 1. Effects of certain antibiotics and bactericides on the growth of Xanthomonas corylina and X. juglandis.

Material	Amount or concentration[a]		Degree of inhibition of X. corylina[b]	Degree of inhibition of X. juglandis[b]
Terramycin	10 µ g.	(in disc)	++	++
Terramycin	100 µ g.	"	+++	+++
Aureomycin	2 µ g.	"	++	++
Aureomycin	20 µ g.	"	+++	+++
Streptomycin	10 µ g.	"	++	++
Streptomycin	100 µ g.	"	+++	+++
Neomycin	10 µ g.	"	+	0
Neomycin	100 µ g.	"	++	0
Polymyxin B	10 µ g.	"	+	0
Polymyxin B	100 µ g.	"	++	0
Chloromycetin	2 µ g.	"	0	0
Chloromycetin	20 µ g.	"	+	0
Bacitracin	2 units	"	0	0
Bacitracin	20 units	"	+	+
Penicillin	1 unit	"	0	0
Penicillin	10 units	"	+	0
Actidione	2.5 mg.	(in cup)	0	0
Actidione	25.0 mg.	"	+	+
Mercuric chloride	1:500	"	++++	++++
Mercuric chloride	1:1000	"	+++	+++
Oxyquinoline benzoate	1:1000	"	++++	++++
Oxyquinoline benzoate	1:10000	"	0	0
Crystal violet	1:1000	"	+++	+++
Crystal violet	1:10000	"	0	0
Puratized Spray[c]	1:800	"	+++	+++
Puratized Spray	1:1600	"	++	++
Sulfanilamide	1:500	"	0	0
Sulfanilamide	1:1000	"	0	0

[a] Units or micrograms (µ g.) per disc and mg. or dilution in cups.
[b] 0=no inhibition; +=less than 1 cm. of inhibition; ++=1 to 2 cm. of inhibition; +++=2 to 3 cm. of inhibition, and ++++=complete inhibition.
[c] 5% phenyl mercury triethanol ammonium lactate.

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OREGON AGRICULTURAL EXPERIMENT STATION AND U. S. BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

AEROPLANE SPRAYING FOR PECAN SCAB CONTROL

John R. Large

This paper presents one year's (1953) results of an experiment to control pecan scab by spraying the trees with five applications of 18X Parzate applied from an aeroplane at about three-week intervals during the period April 15 to July 25.

Pecan scab (Cladosporium effusum (Wint.) Demaree) is the most serious disease of pecans and occurs on several of the paper shell varieties throughout the southern United States. Standard recommendations for control of scab are five or six applications of Bordeaux mixture applied during the summer months from a hydraulic machine[1]. Although this method of application results in commercial control, it has several disadvantages: 1) the high pressure spray machines are so expensive that it is not economical for a pecan grower with a small orchard to own one; 2) in wet seasons the heavy machinery can not be hauled through the orchard; 3) two or three men are needed to operate this spray machine; 4) this hydraulic application is slow and takes several days at a time when other farm work needs to be done. Aeroplane spray application solves most of these problems.

In Georgia and Louisiana pecan insects have been satisfactorily controlled by spraying concentrated insecticides from aeroplanes, but to date only preliminary experiments have been completed on aeroplane spraying to control pecan scab.

Phillips and Cole (1948)[2] tried dusting Moore and Moneymaker pecans from an aeroplane to control pecan scab, foliage diseases and pecan insects. The control by dusting appeared to be very good early in the season. However, by the last of July, which was a wet month, the scab fungus built up to such an extent that the dusted pecan nuts were no better than the untreated ones.

In 1951 a grower near Starke, Florida, sprayed his entire orchard from an aeroplane, making three applications of a concentrated solution of Copper A compound, the last application being made about August 7. On this date nuts on these Success trees were fairly clean and the orchard rated as having very good commercial scab control. In late August the scab disease built up on these trees and the owner applied two more applications of bordeaux mixture with a high pressure ground sprayer. This combination of aeroplane and ground sprayer produced an excellent crop of nuts. An unsprayed Success orchard about a mile away did not produce a crop due to pecan scab[3].

In 1952 a ten-acre plot consisting of a block of 45 Moneymaker and a block of 75 Moore trees was sprayed five times from an aeroplane. The fungicide was concentrated so that enough chemical for 1800 gallons of dilute spray was suspended in 100 gallons of water. The formula was 36 pounds zineb (Parzate) plus 18 quarts of Volk summer oil to 100 gallons. The applications were made April 14, May 15, June 11, July 7, and July 25. Eleven pounds of 25% parathion was added to the May and the two July applications to control leaf casebearer and black aphids.

During the early part of the dry summer of 1952 this aeroplane method of pecan scab control appeared to be equal to the ground application, but after July 15 the scab built up, on the pecan nuts close to the ground, to such an extent that they were considered to be as badly scabbed as unsprayed trees. However, scab counts made August 28 on the Moore trees indicated 87 percent control for the aeroplane spraying.

On this date the scab reading for Moore trees sprayed with bordeaux mixture or zineb (Dithane Z-78) 2-100 applied with the high pressure ground machine was 100 percent marketable nuts. The Moore mean yield and cracking data are shown in Table 1. The unsprayed Moore trees were small so that the yield is not comparable with the other trees. However, the important record is the August scab count, 15 percent marketable nuts.

The scab control reading on August 28 for the nuts on the Moneymaker trees was, for the aeroplane block: 90 percent marketable nuts; for the high pressure ground sprayer: bordeaux mixture 99, bordeaux-ziram split application 100, ziram plus oil 99.6, and zineb (Dithane Z-78) 100 percent marketable nuts. The scab reading for the unsprayed Moneymaker trees was 94 percent marketable nuts. The mean yield and cracking data are shown in Table 2.

---

[1]Phillips, A. M., J. R. Cole, and J. R. Large, 1952. Insects and diseases of the pecan in Florida. Florida Agr. Exp. Sta. Bull. 499: 76.

[2]The author wishes to thank A. M. Phillips of USDA and Fla. Expt. Station and

[3]G. H. Blackmon, Horticulturist and R. H. Sharpe, Associate Horticulturist, Florida Agr. Expt. Station, for this information.

Table 1. Comparison of scab control, yields, and sizes of Moore pecans when sprays were applied with hydraulic and aeroplane sprayers. Monticello, Florida, 1952.

Treatment (6 trees in each)	Type of machine	Number of spray applications	Percent marketable nuts. First 3 scab classes	Mean yield in lbs. per tree	Number nuts per pound	100-nut cracking sample	
						Percent kernel	Percent culls
			8/28/52				
4-1-100 bordeaux mixture, 6-2-100 bordeaux mixture, plus parathion 2-100	High pressure hydraulic	6	100.0	58.0	103	39.7	20.0
Zineb (Dithane Z-78) 2-100 plus oil 1 qt. to 100 plus parathion	High pressure hydraulic	6	100.0	80.7	108	36.2	24.0
Zineb (Parzate) 36-100 plus oil 18 qts. plus parathion 11 lbs.	Aeroplane	5	87.0	51.6	99	35.7	36.0
Not sprayed[a]			15.0	5.0	107	36.5	42.0
Not sprayed[b] -- Cole's trees				66.0	124	33.5	31.0

[a] Small trees were used so the comparison of the yield is not correct, however, the August 28 record indicates the severe infection of scab on the unsprayed trees.

[b] The author wishes to thank J. R. Cole for the data on these unsprayed Moore trees.

Discussion

With aeroplane spraying the upper part of the trees showed much better scab control than the lower portion. This is just the opposite of the usual results where trees have been sprayed from the ground. Many limbs in the top of the trees broke off owing to the growth and weight of the nuts, but many of the nuts on the lower branches were severely scabbed. The three applications of parathion resulted in satisfactory commercial control of pecan insects.

Under the rainfall conditions in 1952, which consisted of drought in June and July and 15 days of rain in August, aeroplane spraying with zineb (Parzate) resulted in commercial control of pecan scab at a cost that was about equal to the cost of other conventional ground spraying, $25 per acre for the five spray applications.

Eight Moneymaker and eight Moore trees were selected at random from the aeroplane sprayed block. The mean yield this season from the eight 35-year old Moneymaker trees was 144 pounds. The mean yield from the eight aeroplane sprayed Moore trees was 52 pounds.

Table 2. Comparison of scab control, yields and sizes of Moneymaker pecans when sprays were applied with hydraulic and aeroplane sprayers. Monticello, Florida, 1952.

Treatment (6 trees in each)	Type of machine	Number of applications	Percent marketable nuts. First 3 scab classes	Mean yield in lbs. per tree	Number nuts per pound	100-nut cracking sample	
						Percent kernel	Percent culls
			8/28/52				
4-1-100 Bordeaux mixture, 6-2-100 Bordeaux mixture Parathion 2-100	Hydraulic	6[a]	99.4	115.0	79	40.7	14.0
Bordeaux-Ziram Split application	Hydraulic	6	100.0	102.0	75	45.0	6.0
Ziram plus oil	Hydraulic	6	99.6	139.0	88	43.6	5.0
Dithane Z-78 plus oil	Hydraulic	6	100.0	131.0	75	41.9	12.0
Parzate 36-100 plus oil 18 qts. plus parathion 11 pounds -100	Aeroplane Concentrate	5	90.0	144.0	71	40.9	7.0
Not sprayed			94.0	134.0	87	38.5	18.0

[a] Spray schedule consisted of one prepollination followed by 5 post-pollination applications. Prepollination date was April 11. Post-pollination dates were May 5, June 5, July 7, July 25 and August 6. The final aeroplane application was made July 25.

The data indicate that the 134 pounds of nuts from the unsprayed Moneymaker trees were smaller and of poorer quality than the nuts from the sprayed trees. On unsprayed Moore trees nuts which remained until harvest were of almost as good quality as those from the sprayed trees. The unsprayed trees were almost defoliated by foliage diseases before the last of September, whereas the sprayed trees held their foliage until frost, about the middle of November. These data confirm earlier observations that pecan scab is more difficult to control on Moore trees than on Moneymaker trees. The pecan scab control obtained by aeroplane spraying with five applications of 18X zineb (parzate) was rather promising in the dry season of 1952, but this may not be true in a normal wet summer. The aeroplane spraying experiments will be continued in 1953.

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CLITOCYBE ROOT ROT - A DISEASE OF ECONOMIC IMPORTANCE IN GEORGIA PEACH ORCHARDS

E. F. Savage, J. H. Weinberger, E. S. Luttrell, and A. S. Rhoads[1]

It is generally recognized that peach trees planted on old orchard sites do not live as long as the trees in the original orchards. This problem has existed for a number of years, and during the late thirties, growers began requesting information relative to its cause and solution.

In 1946, after preliminary survey work, field studies were started at the Georgia Experiment Station to determine the reason for the short life of peach trees planted on these old orchard sites. One of the locations selected for experimental study had been in peaches continuously since 1929. By the end of 1948 it was apparent that the trees replanted on this site were dying from causes that were not immediately determinable. During the spring of 1950 large numbers of trees were below normal in appearance. Digging around the trees to a depth of 6 inches below the ground level showed no injury to the crown. It was then decided to excavate entire trees that were showing injury. At depths ranging from 8 to 14 inches below the soil surface, the bark of the roots was dead, and the mycelium of a fungus was found. At about the same time J. H. Weinberger independently discovered the same fungus present on trees which were planted in 1945 at the U. S. Horticultural Field Laboratory, Fort Valley, Georgia. The only apparent cause for dying of the trees was that the crown and root system were girdled well below the ground line, and a white mycelium was present in the affected areas. Previous to 1952 no fruiting bodies were detected in the field either at Fort Valley or Experiment, and laboratory attempts at identification of the fungus were unsuccessful. During the second and third weeks of September 1952, heavier than normal rainfall was especially favorable for the development of this fungus. On September 17, 1952 sporophores were found in the Georgia Experiment Station orchard by E. F. Savage and identified as Clitocybe tabescens (Fr.) Bres. by E. S. Luttrell. This identification was confirmed by J. H. Miller of the University of Georgia and A. S. Rhoads.

Although its presence had not been realized, this fungus apparently has occurred in Georgia peach orchards for many years and probably has contributed considerably to the loss of trees. There are several reasons for failure to recognize the presence and wide distribution of Clitocybe tabescens. The common cultural practices used by growers of cultivating the soil towards the tree rows, hoeing grass and weeds from the base of the trees, and mounding the trees in the fall for the borer treatment were unfavorable for its fruiting. The dry weather usually prevailing in September and October prevented to a large extent the formation of fruiting bodies of the fungus. Still a third reason was that any injury observed on the peach tree crown was usually ascribed to the chemicals used in borer control or to winter injury. Under these conditions growers and experimental workers alike neither recognized Clitocybe tabescens as causing the death of the tree nor evaluated its importance.

The experimental planting at the Georgia Experiment Station made in 1946 included 98 original plot trees. Of this number 53 had died by September 1952. Of these dead trees about 50 percent died as a result of Clitocybe tabescens, and it would appear that many more died of the same cause although it was not recognized at the time of death. It is interesting to note that at the end of 17 years 95 percent of the trees in the 1929 orchard planted on this site were still living. This is in marked contrast to the 54 percent of the trees dead after six years in the second orchard planted on this site. In this block of trees five clusters of sporophores were found in September 1952. Sporophores were also produced on tree trunks from this orchard when they were placed in a moist chamber. In still another of the Georgia Experiment Station's orchards, Elberta trees budded on Yunnan and Shalil rootstocks were infected with this fungus.

Following the identification of the disease in two widely separated areas, Experiment and Fort Valley, a preliminary survey was made by Savage, Rhoads, and Weinberger of orchards in the principal peach growing sections of Georgia, in order to secure some idea of the distribution of Clitocybe root rot. Many of the orchards in the Fort Valley area of Georgia have been planted with peach trees continuously for 30 to 50 years. Six large orchards inspected in this preliminary survey were found to be infected with Clitocybe tabescens. J. H. Weinberger con-

[1]E. F. Savage, Horticulturist, Georgia Experiment Station, Experiment, Georgia; J. H. Weinberger, Senior Horticulturist, Division of Fruit and Nut Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Fort Valley, Georgia; E. S. Luttrell, Plant Pathologist, Georgia Experiment Station, Experiment, Georgia; A. S. Rhoads, Pathologist, Division of Forest Pathology, Bureau of Plant Industry, Soils, and Agricultural Engineering, Beltsville, Maryland.

tinued the survey and found infected trees in 16 of 18 additional orchards. In some of the orchards in which young replants were dying or dead, C. tabescens was identified on the old roots left in the soil from the preceding tree. It is apparent that after this disease is present in an orchard, there is a gradual build-up of the sources of inoculum. This explains why some two-year-old trees were observed to be succumbing to the attacks of C. tabescens in the Fort Valley area. Weinberger found 30 out of a total of 79 eight-year-old Elberta trees recently pulled to be affected with Clitocybe root rot. Weinberger also reported C. tabescens to have caused the death of trees budded on Tennessee Red Leaf and Bound Brook Red Leaf stocks, both of which are Tennessee Natural selections.

In one orchard in the Middle Georgia peach section in which the grower had reported trouble in getting peach replants to live as early as 1938, there were some interesting findings. A total of 22 dead trees showed the root crown and lateral roots to be infected with Clitocybe tabescens. On 14 of the 22 dead trees the dessicated remains of old sporophores of the fungus were discovered at the base of the trees. On another tree there was a fresh cluster of sporophores. Three additional trees still living were also attacked by the fungus. This orchard had been in peaches continuously for 35 years.

Clitocybe root rot was found in several other orchards in the Middle Georgia peach section. These orchards were old or had been replanted on old orchard sites. Trees in one orchard planted on newly cleared land were found to have this disease. Inspection of trees in young orchards planted on old cotton lands in this same general area failed to show the presence of Clitocybe tabescens.

Root rot caused by Clitocybe tabescens was determined by Rhoads to be of widespread occurrence and destructive in peach orchards in both the Piedmont and Sandhill peach sections of South Carolina.

Rhoads (3) has pointed out that Clitocybe root rot commonly is found in southeastern United States on many different species of plants. Its presence on peach trees was recognized in Oklahoma (5) several years prior to 1901 and in Arkansas (4), Missouri (1), and Florida as early as 1902. It was reported as occurring on apple trees in Georgia (2) in 1920.

The survey made shows that Clitocybe root rot is of general occurrence in peach orchards throughout both the major and minor peach growing sections of Georgia and that it occurs in orchards in both the Piedmont and Sandhill peach sections of South Carolina. In Georgia Clitocybe root rot of peach trees is found mainly on land planted to peaches for periods ranging from 30 to 50 years. The usual history of such orchard sites is a long period of cotton planting previous to the setting of the peach orchard. Only one orchard planted on recently cleared land was observed, and as was expected, Clitocybe root rot was discovered. Yunnan, Shalil, and Tennessee Natural rootstocks are evidently not resistant to this disease. With the gradual build-up of sources of inoculum, peach trees progressively die at an earlier age when they are replanted on old orchard sites.

The prevalence and wide distribution of this disease give at least a partial answer to the question of why peaches will not follow peaches successfully under Georgia conditions. Old peach orchard sites should not be replanted to peaches. It also appears that peaches should not be planted on newly cleared forest land, since apparently there is a relationship between the incidence of this disease and timbered lands.

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PHYTOPHTHORA-INDUCED DISEASES OF AVOCADO IN CENTRAL AMERICA

George A. Zentmyer and Albert S. Muller[1]

A Phytophthora seedling blight of avocado (Persea americana) caused considerable loss in seed beds at the Escuela Agricola Panamericana, Tegucigalpa, Honduras, in 1951, and appeared again in the rainy season of 1952. This note describes this new disease in this area.

Symptoms depend on the stage of growth at which seedlings are affected. When seedlings were invaded at the time of emergence or shortly after the young shoots emerged from the sawdust in which the seed were planted, the shoots turned brown and were killed within a few days. When invasion took place some distance up the stems, after the seedlings had reached a height of 6 to 12 inches, the obvious symptoms were a wilting of the terminal leaves, bending over of the top from the point of invasion, and eventual death of the seedling as the fungus progressed down the stem. The stems were invaded at any location, from the base to the terminal bud. Close inspection revealed definite cankers on the stems, with sporangia abundantly present on the cankered areas during periods of high humidity. Conditions were favorable during rainy periods for dispersal of sporangia in wind-driven rain.

Sporangia on the avocado stems were papillate and ranged in size from 20-30 microns by 27-40 microns. The fungus was probably Phytophthora palmivora Butler. This fungus has been described by Conover (1) as causing a seedling blight of avocado in Florida, and is probably the cause of seedling blight of avocado in the Philippines, referred to by Reinking (2).

The avocado seedlings propagated at the Escuela Agricola Panamericana were seedlings of Persea americana types, from local Honduran markets. In 1951 approximately 80 percent of the seedlings were lost from the Phytophthora blight; in 1952 the disease appeared again in August but affected only a small percentage of the seedlings. Seed are propagated in open beds, beneath a partial canopy of cypress and other cultivated shade trees. Rainfall in this area averages about 45 inches per year, most of which falls from May to October.

In addition to the seedling blight, a number of isolations of Phytophthora cinnamomi were made from roots of avocado trees showing typical root rot symptoms, in the experimental planting at the Escuela Agricola Panamericana. P. cinnamomi was also isolated from roots of a related member of the Lauraceae, Nectandra sinuata. This tree was growing in an experimental planting where trees were affected with Phytophthora root rot; the tree was wilted but the root system had also been damaged by a flood in June 1952, so the poor condition of the tree could not definitely be attributed entirely to P. cinnamomi.

Root cultures were made from a total of 106 trees of various species of Persea in Guatemala, Honduras, Costa Rica, El Salvador, and Mexico in July and August, 1952, in the course of a trip by the senior author to collect Phytophthora root rot-resistant materials for importation into California. Phytophthora cinnamomi was recovered from 18 of these trees in Honduras, Costa Rica, and Mexico. The fungus was found in Honduras at the Escuela Agricola Panamericana, Tegucigalpa, and at the Lancetilla Experiment Station. Several acres of seedling avocado trees were found to be affected with Phytophthora root rot near the town of Orotina, Costa Rica. In Mexico, Phytophthora cinnamomi was recovered from roots of healthy appearing Persea americana and P. schiedeana in the State of Veracruz, from the same area from which the fungus was collected in 1951 (3). Also, near the village of Tenancingo, State of Mexico, an estimated 20,000 avocado seedling trees were affected with Phytophthora root rot; P. cinnamomi was recovered from roots of all trees sampled.

Other diseases of avocado observed were: scab (Sphaceloma perseae) on fruit and branches of Persea americana types in Honduras and Costa Rica, powdery mildew (Oidium sp.) on leaves in Honduras, and several leaf spots, including one caused by Meliola sp. on Persea donnell-smithii in Honduras.

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STRAWBERRY A HOST OF WESTERN ASTER YELLOWS VIRUS

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Frazier and Severin (1) attempted to transmit western aster yellows virus from celery and asters to wild strawberry, Fragaria californica C. & S., with four species of leafhopper vectors. One plant of eight inoculated developed symptoms but efforts to recover the virus to celery or aster were not successful.

During 1950 the senior author again attempted to infect wild strawberry by means of infective aster leafhoppers, Macrosteles fascifrons (Stål) (M. divisus (Uhl.)), bred on aster yellows infected aster or celery plants. Three plants of a runnerless strain of Alpine strawberry, Fragaria vesca L. var. americana Porter, and two each of F. vesca (East Malling clone), F. bracteata Heller (Sonoma clone), and Erodium moschatum L'Her (whitestem filaree) were inoculated by leafhoppers reared on diseased asters. One F. bracteata developed disease symptoms after an incubation period of 27 days while all other inoculated plants remained symptomless. In another test four Alpine strawberry and two whitestem filaree plants were inoculated by leafhoppers reared on infected celery. One Alpine plant developed disease symptoms after an incubation period of 25 days. The virus was recovered from this plant by previously noninfective aster leafhoppers and transmitted to two of two healthy broad-leaved plantain, Plantago major (L.), plants but not to two healthy filaree. Both plantain plants developed symptoms typical of western aster yellows.

The two experimentally infected strawberry plants developed symptoms quite typical of those caused by the aster yellows virus on other host plants. The first evidence of infection consisted of very mild vein clearing and a general chlorosis of the affected leaf. The disease rapidly developed in severity, causing development of adventitious crowns and chlorotic leaves and petioles progressively reduced in size until they became very minute shortly before the plants died. Leaflets exhibited upward or downward cupping and some petioles were twisted or curved and tended to assume a horizontal position.

During 1952 a disease with symptoms suggestive of aster yellows was noted to be common but not abundant in commercial strawberry plantings over widely scattered areas of Central California. In one planting somewhat less than 5 percent of the plants showed the disease but in most fields only an occasional diseased plant was found. For the most part, the disease was found only in plantings of the variety Lassen.

The most diagnostic characteristic of the disease in the field was phyllody of the flowers (Fig. 1) a symptom quite typical of aster yellows. On individual plants, depending on the stage of symptom development, and even on a single inflorescence the degree of phyllody varied from a mere virescence of petals to a condition in which a single flower appeared like a gall or tight rosette of dwarfed green abnormal foliage. On all plants showing phyllody, foliage symptoms were also present and included vein yellowing, general chlorosis of varying shades of pale green, yellowish, or reddish, upward cupping, curvature and twisting of petioles, and an upright brittle stiffness. Plants were sometimes very stunted with greatly reduced leaves and shortened petioles. However, aphid-transmitted strawberry viruses could also be recovered from most plants tested by means of the strawberry aphid Capitophorus fragaefolii (Ckll.). Since foliage symptoms caused by some aphid-borne strawberry viruses may resemble some of the foliage symptoms noted above it is not known to what degree the aphid-borne viruses were responsible for the foliage symptoms observed on the plants with phyllody. Aphid-transmitted viruses are not known to cause phyllody in Fragaria. In the field, plants showing phyllody appear to be self-eliminating as they die within a few weeks or are so obviously abnormal that they are rogued out.

On different occasions and from several localities, plants showing floral phyllody have been transplanted from the field into pots and brought into the greenhouse. In every case these plants have died within two months. At the same time other plants showing only foliage symptoms were similarly transplanted. Only a few of them subsequently developed phyllody and all of these died within a short time afterward.

Attempts were made to recover aster yellows virus from field plants with phyllody symptoms as well as from field plants with normal flowers but with suspicious foliage symptoms. In the recovery trials with plants showing phyllody the procedure followed was to cage non-

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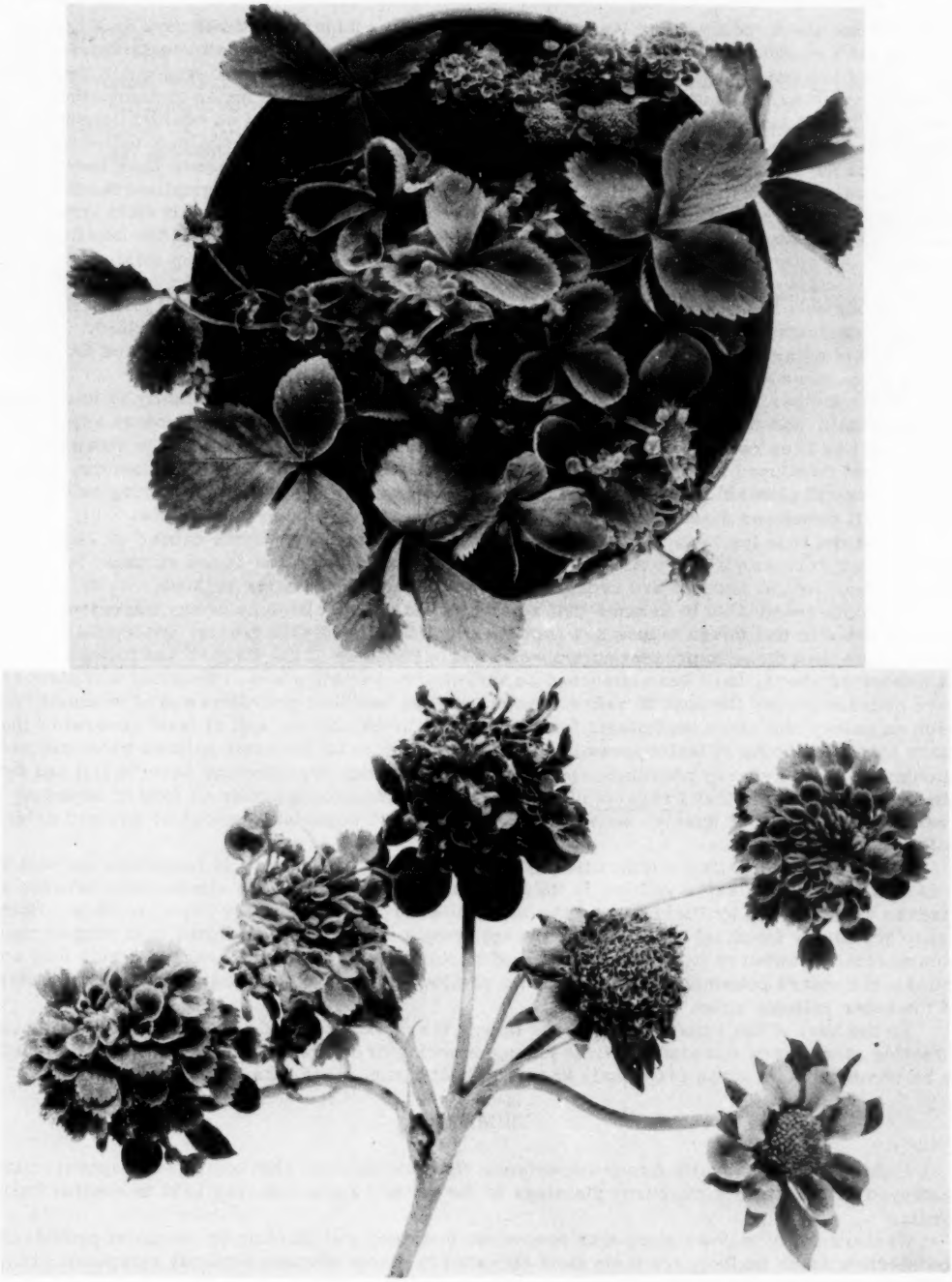

FIGURE 1. Symptoms of western aster yellows on field infected Lassen variety strawberry. The floral virescence and phyllody is diagnostic. Note nearly normal appearing flower at the lower right.

viruliferous stock colony aster leafhoppers on a healthy plantain for three days as a control test on the non-infectivity of the leafhoppers. They were then allowed an acquisition feeding period of from one to seven days, depending upon insect mortality, following which they were transferred to healthy plantain for a period of two to three weeks in order to complete the latent period. Then followed one week on healthy aster and finally a period on healthy Lassen strawberry for five weeks or until all insects were dead. Six field plants were thus tested and all plantain of the first control set remained healthy, proving the leafhoppers to have been non-viruliferous. All plantain and all asters inoculated subsequent to the acquisition feeding period developed typical symptoms of aster yellows, thus demonstrating that the six field strawberry plants showing phyllody were infected with the aster yellows virus. None of the healthy Lassen strawberries in the final set of plants inoculated developed anything but very mild, inconclusive leaf symptoms over a period of six months until moved out of doors for winter dormancy.

A somewhat similar procedure was used in testing eight strawberry field plants showing foliage symptoms thought possibly to be caused by aster yellows but without phyllody. Only plantain and aster were used as test hosts. Aster yellows virus was not recovered from any of the eight strawberries.

It was further attempted to transmit the aster yellows virus experimentally to four Lassen, two Marshall, and three Alpine strawberry plants. For this purpose stock colony aster leafhoppers that had been reared on diseased plantain or aster were used. None of the strawberries have as yet developed any symptoms of disease over a minimum period of six months, although healthy control plantain plants upon which the leafhoppers were fed prior to caging on the strawberries all developed disease, proving the leafhoppers to have been viruliferous.

It has not thus far been possible to distinguish the foliage symptoms caused on strawberry by the aster yellows virus from those produced by some of the aphid-borne viruses. Only the flower symptoms at present are recognized to be diagnostic for aster yellows.

It seems reasonable to assume that aster yellows will not become of any importance on strawberries in California unless new varieties are developed with greater susceptibility to the disease than those in present commercial use. Phyllody of the flowers and foliage symptoms, as described above, have been observed on strawberry varieties and in seedling test plots on rare occasions over the past 20 years. Aster yellows has been prevalent and of economic concern on celery and other host plants for many years in California, and at least several of the more than 20 species of leafhoppers reported to be vectors of the aster yellows virus are not uncommon in strawberry plantings. Greenhouse experience (Frazier and Severin (1)) and field observations indicate that Fragaria plants are not very favorable either as food or breeding hosts of the leafhopper species considered to be the most important vectors of western aster yellows.

Plakidas (2) described a chlorotic phyllody disease of strawberry in Louisiana and stated it to be suggestive of aster yellows in its symptomatology. There are similarities between the disease as described by Plakidas and the western aster yellows disease reported here. Both cause seemingly identical symptoms, are apparently lethal, have a limited host range among commercial strawberry varieties, and are of minor importance in the areas in which they are found. It appears possible that the chlorotic phyllody disease may in fact be caused by a strain of the aster yellows virus.

To the best of the authors' knowledge this is the first record of a leafhopper-borne virus affecting strawberry and also the first virus occurring in nature in commercial strawberries to be identified as a virus previously known on host plants other than Fragaria.

SUMMARY

A disease of apparently minor importance characterized by phylloid flowers appeared in scattered commercial strawberry plantings of the variety Lassen during 1952 in central California.

Western aster yellows virus was recovered to asters and plantain by means of previously noninfective aster leafhoppers from field strawberry plants showing phyllody symptoms, but not from plants showing only suspicious leaf symptoms.

Western aster yellows virus was experimentally transmitted to three of 22 wild strawberry plants and the virus recovered to plantain from one of the three infected strawberries. Attempts to infect ten Lassen and two Marshall plants were not successful.

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VIRUS YELLOWS OF TABLE BEETS IN THE PACIFIC NORTHWEST

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Table beet seed (Beta vulgaris L.) production has been an agricultural specialty in the Skagit Valley of northwestern Washington for nearly half a century. As early as 1940 a disease which causes yellowing and foliar necrosis was observed in this area but only recently was it shown to be incited by a virus. Nutritional deficiencies were considered previously as possible causes. A similar yellowing has been recognized in Europe for many years as "virus yellows", a serious disease of sugar beets, red-beets, and forage beets (mangel wurzel) (3, 4, 9). Coons (2) recently showed the similarity between virus yellows of Europe and a now widespread yellows disease of sugar and table beets in the United States. Virus yellows is distinct from mosaic reported by Jonas (5) and Pound (8) as severe in seed beets in the Puget Sound region.

The two-year cycle of beets grown for seed and the methods of culture used in this area have tended to increase the severity of virus yellows. Seed is sown in beds during June. In late fall the roots are taken up and placed in pit storage for the winter. The following spring they are transplanted to seed rows, and during August and September seed is harvested. Seed is produced by farmers for seed contractors who supply stock seed from which the farmers grow the roots (stecklings) or supply the stecklings themselves.

This paper is a preliminary report of a study of the occurrence, symptoms, and the insect relations of the virus causing virus yellows in table beet seed crops in the Pacific Northwest.

SYMPTOMS OF THE DISEASE: On stecklings transplanted for seed production in March or April, symptoms of virus yellows are not clearly distinguishable until June or July. The first noticeable symptoms are expressed in older leaves of infected plants, which become chlorotic, notably thickened, leathery in texture, and brittle. Symptoms are not apparent in young leaves at first and no foliar patterns or pronounced vein clearing has been observed. Infected seed plants often develop excessive anthocyanin pigment. Chlorotic leaves become orange-yellow to bronze with conspicuous interveinal, reddish spots which enlarge, often coalesce, and become necrotic (Fig. 1). Chlorosis and necrosis progress until in the final stage, about the time seed plants are maturing, the plant shows a general decline and has abnormally red or yellow, often necrotic, foliage. Affected seed plants have poor root growth and are easily uprooted in a moderate wind. Seed from infected plants is smaller and has less vitality than seed from uninfected ones (3).

Mangel, sugar beet (B. vulgaris), and chard (B. vulgaris var. cicla L.), as well as table beet, are affected and characteristically develop similar symptoms such as yellowing of leaf blades, especially at the tips, and thickening and brittleness of the leaf tissues. Infected spinach (Spinacia oleracea L.) seed plants are yellow and stunted. Susceptible weed hosts have not been investigated in the Pacific Northwest. In England the virus infects a number of chenopodiaceous weeds among which is lamb's-quarters (Chenopodium album L.), a common weed (3).

Typical symptoms of the disease are not expressed in the greenhouse under conditions of low light intensity and unfavorable growing conditions such as prevail during the winter. Even under ideal conditions during summer, inoculated plants in the greenhouse frequently show only yellowed outer leaves which become wilted instead of brittle.

TRANSMISSION OF THE VIRUS: Transmission of the yellows virus is readily obtained with the green peach aphid (Myzus persicae (Sulz.))(3, 4, 7, 10, 12). Although in the Pacific Northwest this vector is seldom present in great numbers either in root beds or in seed fields, it is believed to be of primary importance in the transmission of the virus. The virus persists in the aphid for an indefinite period (11). Neither the black bean aphid (Aphis fabae Scop.) nor the beet root aphid (Prociphilus betae Doane) is considered an important vector, but the latter has not been adequately tested (10). The black bean aphid is the most common insect in beet seed fields.

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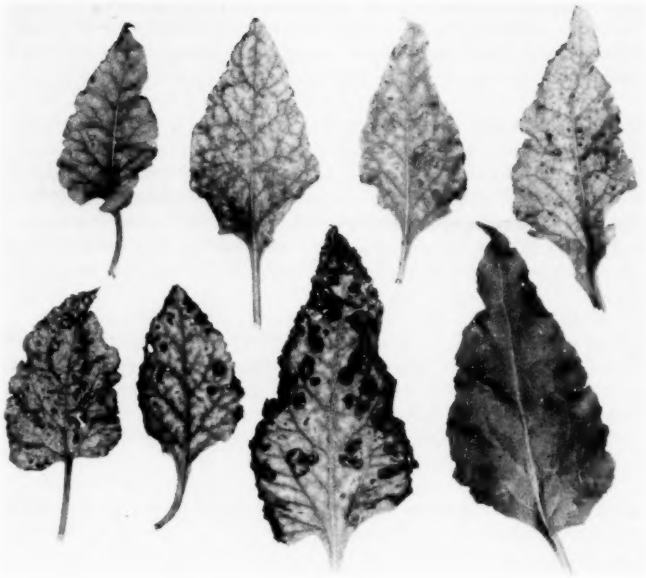

FIGURE 1. Leaves taken from a table beet seed plant infected with virus yellows showing progressive stages of chlorosis and necrosis. Leaf from healthy plant at lower right. (Courtesy M. A. Watson)

Most European investigators have not regarded mechanical inoculations as serious in transmission of the yellows virus. Kassanis (6) reported that mechanical inoculations with the virus resulted in about 10 percent of the plants becoming infected. More recently, Coons (2), using carborundum as an abrasive, was able to show a very low percentage of successful juice inoculations. Although mechanical transmission is possible under experimental conditions, cultural practices in handling beet roots, planting, and cultivation are not thought to account for the presence or distribution of the disease in seed fields.

European investigators who have studied virus yellows have found no evidence that the virus was transmitted through the seed. In a preliminary note Clinch and Loughnane (1) described a yellowing disease in a breeder's "pure line" of sugar beet (family 41), reported to be transmissible through the seed. It was shown later by Watson, et al. (13) that this report of seed transmission of virus yellows was inconclusive and has not been confirmed. No evidence of seed transmission in table beet seed has been observed during investigations of the disease in northwestern Washington.

CONTROL: Cultural methods followed in the Puget Sound section for production of beet, mangel, and Swiss chard have been ideal for perpetuation of virus yellows. Root beds have been planted indiscriminately in relation to maturing seed fields and often adjacent to them. Aphid migration from seed fields to root beds has resulted in very widespread infection of seedling plants. Biennial seed plants such as beet, mangel, and Swiss chard frequently have shown 100 percent infection. The concentration of these seed fields in the Skagit Valley perpetuates the source of virus.

Recommendations to isolate root and plant beds in areas distantly removed from diseased seed fields have resulted in excellent control of beet mosaic (8) and cabbage mosaic (7), respectively. Formerly mosaic was the most important disease in beet and cabbage seed fields in the Puget Sound section. It is probable that, because of the persistence of yellows virus in aphid vectors and the widespread culture of biennial seed crops, isolation of root beds alone has not effected adequate control of virus yellows. It is essential that aphids be controlled in isolated root beds and during the early stages of growth in seed fields. Several years' testing of DDT, BHC, parathion, malathion, Metacide, and Systox[2] for controlling the vectors in root beds in the vicinity of infected seed plants failed to give complete control. Only Systox, a

[2]Systox has not been approved for use on any food crop, to date, and it is not recommended for use on steckling beds. All these insecticides are poisons and must be handled with prescribed preeaution.

systemic insecticide, gave appreciable control on beet stecklings grown in the general vicinity of the seed-producing area. In root beds isolated from the seed-producing area, fair to good control has resulted with all insecticides used, the effectiveness depending largely on the frequency and thoroughness of application.

DISCUSSION: At present, virus yellows is the most important disease of beet seed plants in the Puget Sound section of Washington. The disease apparently has been present for many years in this area. In 1931 Jones (5) described mosaic in seed beets in northwestern Washington. He stated that certain seed fields were 100 percent infected at that time but did not mention any other virus disorder except curly-top. Pound (8) in 1946 stated that mosaic was the most important disease of seed beets in the Puget Sound section and did not mention other disorders. Records since 1940 show that virus yellows has existed along with mosaic but has not been distinguished therefrom.

Virus yellows is also present in Michigan, Colorado, Utah, Oregon, and California (2). Observations have led to the assumption that yellows is most severe in the Puget Sound section, probably because of the concentration of susceptible vegetable seed plants. In Europe several related viruses which cause a multiplicity of symptoms in beets have been described (1). This multiplicity has caused European workers to refer to "mild" yellows and "severe" yellows. European workers who have observed yellows in the United States as described in this paper have concluded that only the "mild" yellows type is present.

The biennial cycle of the various vegetable seed crops grown in the Puget Sound region necessitates the strict adoption of certain cultural practices if virus disorders are to be avoided. Either aphid vectors must be effectively controlled in root beds grown in the vicinity of seed crops or steckling plants must be grown in areas free from viruliferous aphids. It has not been possible to control aphid vectors sufficiently to prevent widespread virus infections in the seed-producing area, probably because of the migration of viruliferous aphids from home gardens, weed suscepts, volunteer beet plants, and infected seed crops. Consequently, it has been necessary to grow stecklings in sections well isolated from seed fields and to control migratory aphids. In recent years it has become difficult to locate isolated areas where susceptible seed crops have not been grown and where the virus is not present. Only complete cooperation of all seed growers and contractors in the isolation of steckling plant beds of all susceptible biennial plants, along with aphid control, can control virus yellows.

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NOORDAM'S B VIRUS OF CHRYSANTHEMUM DETECTED IN THE UNITED STATES

Philip Brierley and Floyd F. Smith

In 1952 Noordam (6) described from the Netherlands three viruses from chrysanthemum infectious to solanaceous plants, namely a chrysanthemum strain of cucumber mosaic virus and chrysanthemum viruses B and C. The virus which he classed as Cucumis virus 1 strain chrysanthemum is evidently the one called tomato aspermy virus by English (2) and American (3, 4) workers. The aspermy virus is prevalent in western Europe and is believed to be increasingly common in the United States, although only six separate identifications are known to us. The aspermy virus is manually transmitted to tobacco, petunia, and Physalis angulata, producing systemic symptoms in all.

Noordam found that his B virus produced yellow local lesions in petunia about three weeks after inoculation. No systemic symptoms developed in petunia, and no infection resulted in tobacco or tomato. Seven of 31 plant species that Noordam inoculated, namely snapdragon, calendula, China aster, Helichrysum bracteatum, Nicotiana glutinosa, petunia, and chrysanthemum, proved susceptible.

At Beltsville we first used petunia as an index plant for chrysanthemum viruses in February 1953, when we tested 60 chrysanthemum stocks of known and unknown virus content on petunia, tobacco, and Physalis angulata.

Tomato aspermy appeared as systemic veining and mottling, recognizable in petunia after six days and in tobacco and Physalis after eight days, from only seven stocks. Sources of the aspermy virus were our culture of this virus in the chrysanthemum variety Nightingale, various transfers from this, and also the garden chrysanthemum Pepita received from Michigan. Petunia proved as sensitive as tobacco or Physalis in reacting to the aspermy virus.

After 14 to 21 days solid yellow local lesions, usually 2 to 3 mm. in diameter at first and characteristic of Noordam's B virus, appeared in petunia sap-inoculated from 17 chrysanthemum stocks. Typically no systemic effects followed in petunia but the local lesions sometimes extended slightly along veins and in one plant a single branch was systemically invaded. The B virus was subcultured from this systemically invaded branch, but not from the symptomless upper leaves of these petunias. No symptoms appeared in tobacco or Physalis from these 17 inoculations and the B virus was not recovered from these plants. Chrysanthemums yielding these B local lesions in petunia were Crystal Valencia (mosaic), Good News (mosaic), Blanche (mosaic), Blanche (crinkle), Matador (mosaic), Paris daisy (symptomless) (Chrysanthemum frutescens), the garden chrysanthemums Lavender Lady, Lee Powell, Major Cushion, Red Velvet, Remembrance, Roza, and transfers from these independent samples.

The distinctive local reaction in petunia, the time of appearance of these symptoms, and the failure to infect tobacco indicate that the virus is Noordam's B virus. This virus is present in the Nightingale stock that we have maintained as a source of aspermy virus and in Good News and Blanche stocks maintained as sources of Keller's Q virus (5), and it occurs also independently of these. The presence of this additional virus in our stock sources of aspermy (3, 4) and Q mosaic (3, 4) viruses calls for reevaluation of our data on these two viruses. Since the B virus was not detected in our stock sources of stunt virus (3, 4) in Mistletoe or of rosette virus (3) in Ivory Seagull, these viruses are not confused with B virus.

The symptoms produced in Good News and presumably in other chrysanthemum varieties also inoculated by grafting with the Nightingale variety are due to the B virus rather than to the aspermy virus. Inoculation from this source by grafting, by the aphids Macrosiphoniella sanborni (Gill.), Myzus persicae (Sulz.), M. solani (Kltb.), and Rhopalosiphum rufomaculatum (Wilson), and by sap, all yielded the same diffuse, often patchy, dull-yellowish mottling without leaf distortion in Good News. Sap inoculation with the aspermy virus from tobacco or from Physalis angulata produced no symptoms in the varieties Dynamo, Good News, Horizon, Mary Garden, Pandora or Yellow Shasta, but the aspermy virus was recovered from these. The lack of correlation between expression of symptoms in chrysanthemums and recovery of the aspermy virus from them (Table 1) has long puzzled us. It is now clear that the B virus is present but was previously unsuspected in our Nightingale stock and that the B virus is responsible for symptoms in chrysanthemum that we have ascribed to aspermy virus. Moreover other stocks of aspermy virus that have come to our attention, namely Delia and Mayford Pink from England and an unnamed variety from Denmark, also carried the B virus together with aspermy virus, inasmuch as these produced the same symptoms in Good News and other chrysanthemum varieties as did the Nightingale stock. B virus symptoms in Good News developed 30 to 40 days

Table 1. Symptom expression and detection of viruses in various chrysanthemums infected with aspermy virus (TAV), with Noordam's B virus (B), or with both.

Source	Date inoculated (1952)	Tobaco test (3-28-52)	Chrysanthemum symptoms (10-2-52)	Physalis test (10-2-52)	Chrysanthemum symptoms (12-5-52)	Physalis test (12-5-52)	Chrysanthemum symptoms (2-10-53)	Petunia test (2-10-53)	Tobacco test (2-10-53)
Graft inoculations from Nightingale infected with TAV and B:									
Alpine	2-29		0	TAV					
Blazing Gold	1-14	TAV	0	TAV					
Dynamo	5-13		0	TAV					
Golden Harvest	2-29		0	TAV					
Good News	1-16	TAV	B	TAV					
Lillian Doty	2-29		B	TAV					
Pandora	5-13		B	TAV					
Rusticon	1-16	0	B	TAV					
Seagull	2-29		0	TAV					
Silver Sheen	2-29		0	TAV					
Golden Curry	2-29		B	0	B	TAV			
Vibrant	2-29		B	0	B	TAV			
Charmer	2-29		B	0	B	0	B	TAV	TAV
Mary MacArthur	2-29		0	TAV	0	TAV	0	TAV	TAV
Mistletoe	1-14	TAV	B	0	B	0	B	B	0
Transfers from Nightingale to Good News by aphids:									
Myzus solani	3-14	TAV	B	0	B	0	B	TAV	TAV
Rhopalosiphum rufomaculatum	3-14		B	0	B	0	B	B	0
Nightingale (source)	--	TAV	0	TAV	B	TAV	0	TAV	TAV
Roza (source)	--		0	0	0	0	0	B	0
Good News grafted with Roza	6-11		B	0	B	0	B	B	0

after sap inoculation from affected petunia.

Inasmuch as aspermy virus is infectious to tobacco, tomato, and Physalis, but B virus is not, experimental transmissions to or from these test plants deal with aspermy only. Thus, our data on host range and properties of aspermy virus are unaffected by detection of B virus in Nightingale. Aspermy virus is damaging to tomato, tobacco, lettuce, spinach, pepper, and a number of ornamental plants. It is doubtful whether it causes any significant damage in chrysanthemum, for it is symptomless in Good News and in other sorts. The conspicuous flower distortion frequently ascribed to aspermy virus by European writers (1, 6, 7) has never appeared in our cultures. All except Roza of the 17 varieties listed in Table 1 have been flowered at least once when affected with aspermy virus plus B virus. Several of these have been flowered in summer and in winter; none has ever showed distortion of the flowers from the combination of these two viruses. It seems likely than an additional component virus, not yet recognized in the United States, is concerned in the distortion of chrysanthemum flowers.

Because aspermy is damaging to vegetable crops and is not known to be generally prevalent in the United States, some propagators wish to exercise special care to avoid disseminating it.

Table 2. Transmission of tomato aspermy virus (TAV) and of Noordam's B virus (B) by aphids.

Aphid species	Source plant	Test plant	Plants infected[a]	Virus transmitted
Anuraphis helichrysi	Chrysanthemum[b]	Chrysanthemum[c]	0/10	--
Aphis gossypii	Chrysanthemum	Chrysanthemum	0/13	--
	Chrysanthemum	Tobacco[d]	0/10	--
Macrosiphoniella sanborni	Chrysanthemum	Chrysanthemum	1/10	B
	Chrysanthemum	Chrysanthemum	3/10	B
	Chrysanthemum	Tobacco	2/10	TAV
Myzus persicae	Chrysanthemum	Chrysanthemum	2/10	B
	Chrysanthemum	Tobacco	8/10	TAV
	Chrysanthemum	Tobacco	3/5	TAV
	Chrysanthemum	Tomato[e]	5/10	TAV
	Tobacco	Tobacco	2/5	TAV
	Tobacco	Tomato	5/10	TAV
	Tomato	Tomato	5/10	TAV
Myzus solani	Chrysanthemum	Chrysanthemum	2/10	B
	Chrysanthemum	Tobacco	5/5	TAV
	Tobacco	Tobacco	3/5	TAV
	Tomato	Tomato	10/10	TAV
	Tomato	Tomato	10/10	TAV
Rhopalosiphum rufomaculatum	Chrysanthemum	Chrysanthemum	0/10	--
	Chrysanthemum	Chrysanthemum	5/10	B

[a] Number infected over number exposed.
[b] Nightingale variety as source.
[c] Good News variety as test plant.
[d] Turkish tobacco var. Samsun.
[e] Variety Marglobe.

The data in Table 1 show that the index test on tobacco or on Physalis is not consistently reliable for indicating presence of aspermy virus. Some varieties, such as Nightingale and Mary MacArthur, yielded this virus whenever tested. Others, such as the first 10 varieties listed in Table 1, yielded aspermy when tested in October 1952, and were not sampled again. Still others such as Golden Curry, Vibrant, and Charmer yielded no aspermy virus until the second or third test, 9 to 11 months after graft inoculation. The difficulty in detecting the aspermy virus does not lie in the test plants, for tobacco, Physalis, and petunia appear to be adequately and about equally sensitive. Apparently the aspermy virus is slow and erratic in invading chrysanthemum systemically.

Experiments with aphid transmission from the Nightingale chrysanthemum, now known to carry B and aspermy viruses, yield data on vectors of both viruses. The mottle pattern in Good News shows presence of B virus, and reaction in tobacco or tomato shows presence of aspermy virus. Thus, the date in Table 2 show that B virus was carried by Macrosiphoniella sanborni, Myzus persicae, M. solani, and Rhopalosiphum rufomaculatum; aspermy virus was carried by the first three of these species but was not shown to be transmitted by the last. No evidence was found that Anuraphis helichrysi (Kltb.) or Aphis gossypii Glov. transmitted either virus. All these tests were designed to detect nonpersistent transmission.

The B virus is common in United States as well as in Europe. It has been detected repeatedly in graft-indexing American chrysanthemum varieties, but has been construed as a mild strain of Keller's Q virus (5), which we have termed chrysanthemum mosaic virus (3).

Since the type stocks of Q virus in Blanche and Good News cause the B virus reaction in petunia, we may assume that B virus is a mild form of Q virus, or that Q virus is a complex with B virus one of the constituents. Evidence is insufficient to decide between these two hypotheses at present, but the second is favored.

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BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING AND BUREAU OF ENTOMOLOGY AND PLANT QUARANTINE

MALFORMATION OF CARNATION BLOSSOMS CAUSED BY ASTER YELLOWS VIRUS[1]

W. D. Thomas, Jr.

A malformation of the blossoms of the carnation variety Hercules has been observed recently in commercial greenhouses in the Denver and Colorado Springs areas in Colorado. The plants on which the malformation occurred showed symptoms of streak. It has been demonstrated that streak is caused by the aster yellows virus[2, 3]. There has been, however, no description of blossom damage by this virus.

The malformation is first expressed in unopened buds by pinched and tight calices (Fig. 1A) on which the normal tipped peak (Fig. 1B) is missing. Upon opening, the calyx appears to have been gnawed, the segment tips being indistinct (Fig. 1C, E). Its length is approximately one-half that of a normal calyx (Fig. 1D, F). The affected blossoms, themselves, are somewhat stunted.

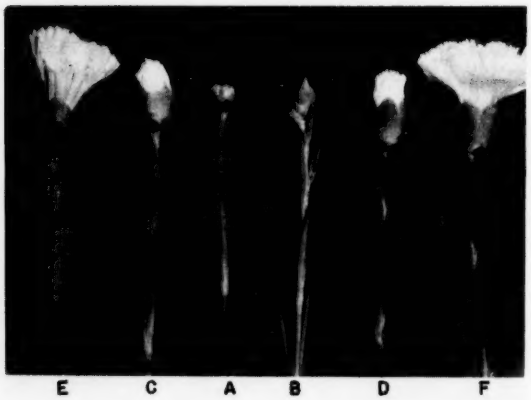

FIGURE 1. Illustration of effect of aster yellows virus infection on carnation buds and flowers. A, C, E -- infected; B, D, F -- normal. Variety Hercules.

Approach grafts between ten affected and ten normal Hercules plants showing no streak symptoms resulted in the production of bud and blossom symptoms in each case when average daytime greenhouse temperatures exceeded 80° F. Streak symptoms developed on all plants showing bud malformation. Check plants grafted to normal plants showed no symptoms.

Infected plants produced malformed blossoms only as long as daytime greenhouse temperatures remained abnormally high. Bud initials formed after daytime temperatures were decreased to 70° F. produced normal blossoms.

When ten normal plants each of White Sim and Miller's Yellow carnations were grafted to streak-infected Hercules plants, no malformation resulted at either 70° or 80° F. daytime temperatures. It is concluded, therefore, that this bud malformation was due to a varietal expression of aster yellows, influenced by high temperatures.

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[1] Published with the approval of the Director, Colorado Agricultural Experiment Station, as Scientific Journal Series No. 407.

[2] W. D. Thomas, Jr. 1949. Hosts of carnation viruses other than carnation. Carn. Craft, 11: 6-8.

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TOPPLE, A NEW DISEASE OF GLADIOLUS IN WESTERN NORTH CAROLINA

Harry C. Fink[1]

Gladiolus grown in Western North Carolina in the 1952 season was subject to a topple disease (Figure 1). Growers report that high percentages of cut spikes of varieties New Europe, Corona, Snow Princess, and Picardy were affected. This disease appeared after the flowers were harvested and was never observed in the field. The characteristic symptom was a breaking of the flower spike and toppling of the flowers. The topple-break occurred anywhere on the spike, but usually about one-quarter inch above a point of flower attachment. No lesion or water-soaked area marked the place where topple might occur; the opening of the flowers on the distal end of the spike seemed to add weight enough to cause the stem to bend over and the tissue to break. Breaks in the epidermis were rare and usually occurred only after the toppled stems were disturbed. The area near the break became water-soaked in two to three days after topple occurred and a strong sour odor became apparent shortly thereafter. No exudate was observed at any time before or after topple occurred.

FIGURE 1. Gladiolus affected with topple. Left and right, variety New Europe. Center, variety Corona.

Tissue plantings from 50 newly toppled stems on P. D. A. plates indicated that neither bacteria nor fungi are associated with the disorder. Platings made four days after toppling from 40 spikes which had become water-soaked and had developed an extremely sour odor yielded 18 colonies of peritrichous bacteria. These bacteria are similar to Erwinia carotovora and are assumed to be secondary soft rotting organisms.

Climatological conditions during the time topple occurred were near normal except for rainfall, which was extremely low in July, normal in August, and moderately low in September and October. Since the topple disease occurred throughout the season, July to October, it seems that climatological conditions influenced the disease very little.

The similarity of this disease to the well known topple disease of tulips, which has been corrected with calcium supplements, suggested that the same factor might be concerned here. To test this hypothesis basic slag, hydrated lime, dolomitic limestone and landplaster were applied to the soil at rates giving 500 pounds of calcium per acre. Calcium nitrate was applied as a 2 percent foliage spray at the rate of 54 gallons per acre. The materials were applied to three 50-foot replications each on varieties New Europe and Corona approximately two weeks before bloom.

During the flowering period spikes were cut every two days from each plot, placed in containers with water, and held at room temperature. Point of topple on the spike and dates of topple were recorded for twelve days for each cutting. The numbers of spikes affected in each variety and for each treatment are presented in Table 1.

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Table 1. Incidence of topple on gladiolus spikes after treatment with five sources of calcium compared to no treatment.

Treatment[a]	Flower spikes harvested		Flower spikes affected with topple		Total percentage of topple
	New Europe	Corona	New Europe	Corona	
	No.	No.	No.	No.	%
1. Basic Slag	71	21	41	8	53.26
2. Hydrated lime	72	22	29	11	42.55
3. Dolomitic limestone	72	19	28	10	41.07
4. Land Plaster	71	18	35	11	51.57
5. Calcium Nitrate	69	22	13	4	18.68
6. No Treatment	73	17	50	10	66.66

[a] All materials were applied to soil except calcium nitrate which was applied as a foliar spray.

Only 18.7 percent of the flowers sprayed with 2 percent calcium nitrate toppled, while 67 percent of those in the untreated checks toppled. Soil treatments had little or no effect upon the 1952 crop. Tests will be continued in 1953 to determine if calcium deficiency results in flower topple of gladiolus.

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THE 1952 EPIPHYTOTIC OF A SOIL-BORNE WHEAT MOSAIC IN KANSAS[1]

H. Fellows, W. H. Sill, Jr., and C. L. King[2]

Beginning in the early spring of 1952 a severe yellowing[3] of wheat was reported from many counties of southeastern and south central Kansas. Numerous affected plants sent to Kansas State College uniformly showed extreme yellowing, particularly at the tips and around the leaf edges, along with mosaic-like mottling of many leaves. The yellowing symptoms largely disappeared, and the mosaic mottling became less severe when these plants were grown in a warm greenhouse. A few developing leaves continued to show greenish to yellow mosaic-like mottling and streaking. The malady looked like a virus disease, but attempts to inoculate other wheat plants manually in a warm greenhouse with extracts from infected plants were uniformly unsuccessful.

A survey of all affected fields that had been reported, and many others, indicated that the malady was present throughout the area. Affected field spots looked yellow when viewed from a distance. The affected areas ranged in size from a few feet in diameter to huge irregular sections in a field. A few fields appeared to be uniformly affected throughout. The condition appeared on both high and low lands, flooded or nonflooded, and in wheat grown continuously or in rotations with other crops. Affected areas apparently were not associated with soil fertility differences since they often extended through both fertile and unfertile areas. Poorly drained low areas and areas that had been flooded appeared to be affected more severely, but this situation also was not always consistent. The field symptoms were very similar to those described by McKinney (2) and Koehler et al. (1) for soil-borne wheat mosaic.

A soil-borne wheat mosaic has been reported from Kansas (5). In the 1949-50 season a soil-borne mosaic was found in eleven places by two of the writers (Fellows and King), mostly along the Kansas River in Douglas and Wabaunsee Counties and in the Frankfort area in Marshall County. It was present also in Shawnee, Jefferson, Jackson, and Brown Counties.

Only three affected fields were found in the 1950-51 season, one in the Frankfort area again and two near Hoyt in Jackson County. Little importance was attached to the disease until the 1951-52 season because of this sporadic and local occurrence.

Soil samples from certain fields in which the wheat showed mosaic symptoms were tested by H. H. McKinney (5) over a series of years, including the season of 1951-1952, and found to be infested with a soil-borne virus. In several of the fields tested the diseased condition reappeared each year in the same locations. These symptoms were identical with those seen over large areas in 1951 and 1952. Several surveys were made during the growing season up to harvesttime in which the tested infested fields and many others were examined. As shown in Figure 1, the outbreak in 1952 was confined largely to the eastern third of the State and was generally more severe in the southcentral and southeastern counties. The disease was observed in 36 counties but probably was present in others. Counties that were most severely affected were: Pottawatomie, Wabaunsee, Harvey, Sumner, Cowley, Montgomery, Labette, Cherokee, Wilson, Greenwood, Johnson, Douglas, Franklin, Neosho, Shawnee, Lyon, and Sedgwick.

Developing symptoms were observed in the field as wheat growth advanced. In general, they were similar to those described for soil-borne wheat mosaic by Koehler et al. (1) and earlier by McKinney et al. (3), except that no rosetting was seen at any time. Experiments are being continued to determine the identity of the virus or viruses involved. As spring temperatures increased and during the active period of vegetative growth, symptom severity declined in all areas resulting in remarkable recovery in most cases. The yellowing largely disappeared, but infected plants in most fields continued to be stunted, and mosaic mottling

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[3]Eastern Kansas records show that since 1925 (and possibly before) there has been periodic "early spring yellowing" of wheat which later in the season disappeared. This usually has been attributed to nutritional difficulties, particularly to nitrogen deficiency; however, in retrospect the senior author feels that some of these cases may have been the soil-borne mosaic discussed here. General field symptoms were often identical. Symptoms became less severe with the advancing season, often disappearing entirely. Also stunting of mature plants with some reduction of head size was noticed.

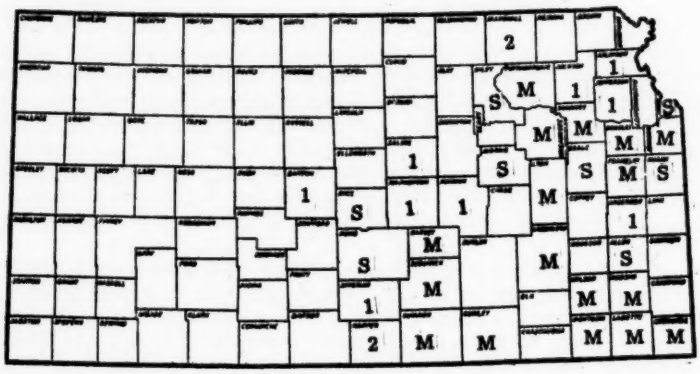

FIGURE 1. Kansas counties in which a soil-borne wheat mosaic was present during the spring of 1952. Numbers indicate the number of infected fields found in the county. S = several infected fields. M = many infected fields.

persisted in some cases. After heading there was a marked reduction in mosaic mottling, so much so that often it was difficult or impossible to find plants showing this symptom even in known infected spots that had been observed periodically. Affected plants often showed delayed heading and maturation in addition to the stunting. Heads on affected plants typically were somewhat shorter than normal. In some fields that were infected severely in early spring, few or none of the late symptoms were noted and recovery was apparently complete. In one area in Johnson County that was not seen until late in the season there was severe stunting, decreased stand, and, consequently, marked decrease in yield. Other diseases, including take-all (Ophiobolus graminis), were present and might have contributed considerably to the losses.

On the whole losses from the mosaic disease were relatively light. No apparent losses occurred in many infected fields. Sample rod-row yields were recorded in fields in which there was stunting, slightly smaller heads and, in some cases, fewer tillers. The wheat varieties, Blue Jacket, Pawnee, or Triumph, were grown in the fields checked. Yields from affected areas ranged from 8 to 13 percent of those from random healthy areas of the same fields. This yield reduction is similar to that reported by Koehler et al. (1) for the Triumph and Pawnee varieties infected with the soil-borne wheat mosaic in Illinois. Both Triumph and Pawnee were reported to be moderately resistant. In Kansas in 1952 Pawnee typically showed severe mosaic mottling in the early spring but recovered rapidly during the latter part of the growing period.

The single year's observations suggest that many of the wheat varieties commonly grown in eastern Kansas are at least moderately resistant to this soil-borne wheat mosaic, but this can be determined only by tests in heavily infested areas.

Summary

An epiphytotic of a soil-borne wheat virus disease in the eastern counties of Kansas is reported. Although not a new disease in the State, this is its first known widespread occurrence of economic significance. It appears to be similar to the disease reported from Oklahoma (6).

It is uncertain whether this is the same disease as the mosaic rosette occurring farther east. Although other characteristics checked are similar, no rosetting has been seen thus far, even on varieties which typically gave this symptom when grown in infested soils in Illinois, and eastward. Work is being continued here in an attempt to identify the virus or viruses involved with certainty.

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U. S. BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AND KANSAS STATE COLLEGE

YIELD REDUCTIONS IN GLACIER BARLEY ASSOCIATED WITH A VIRUS INFECTION[1]

R. F. Eslick[2]

Various foliage symptoms have been noted in barley from time to time and those from which no consistent organisms have been isolated have been attributed to abnormal physiological conditions in the plants. This has given rise to the use of the terms "physiological leaf spot", "physiological stripe", "false stripe", and "brown spot", (1, 2 p. 21). The cause has been presumed to be environmental or hereditary rather than pathogenic.

More recently McKinney (3) has described the symptoms of a seed-borne virus that caused the typical brown false stripe leaf markings in association with chlorotic mosaic mottling and striping in barley. These symptoms have appeared in varying degrees in the Glacier barley plots reported here. McKinney (4) has observed these symptoms in Glacier barley plants grown from the same seed sources used in this study. He has also isolated virus from Glacier plants that expressed no visible signs of stripe mosaic virus (false stripe).

Materials and Methods

Two seed sources were compared in this study. The seed source designated as "diseased" originated from the 1947 yield nursery grown at Bozeman. At the close of this study, in a 37-plant sample grown from the 1951 seed McKinney (4) found 89 percent of the plants to be virus-infected. The lot designated as "disease-free" was obtained from a certified Glacier seed grower's 1947 crop. McKinney (4) found that 5 percent of the plants from this source, grown from 1951 seed, were virus-infected.

In all years, except 1951, when 1949 seed was used, seed grown the previous year was planted.

The yield comparisons reported were obtained by standard nursery trial techniques. Yield determinations were based on from 3 to 6 replications of 16-square-foot harvest areas. The data were analyzed by appropriate analysis of variance methods.

Results

Symptom expression was variable for the years of this study. The foliage symptoms were severe in only one year, 1948, moderate in 1947, 1950, and 1951, and nearly non-existent in 1949.

Yields of the two seed sources are reported in Table 1. In every case the difference in yield was shown to be statistically significant, except for 1947 when the analysis of variance method was not applicable. For 1947 it was necessary to calculate a theoretical yield of "disease-free" Glacier by the missing plot method. This calculation was based on five years' (1945 to 1949) yield data for the varieties Glacier, Compana, and Titan.

It will be noted that the yield reduction occurred every year, and for the five-year period amounted to an average loss of 31 percent. The yield loss was associated with the presence of a high proportion of virus-infected plants.

It is standard procedure to attempt disease control by seed treatment. Accordingly, treated and non-treated seed from both sources were grown for the three years 1949 to 1951. Yields are reported in Table 2.

Seed treatment has no statistically significant effect on yield from either source in any one year.

Summary and Discussion

The effect on yield of a seed-borne barley disease heretofore not considered serious was determined over a five-year period, 1947 to 1951, in the variety Glacier. The average yield reduction amounted to 31 percent. Foliage symptom expression was not in proportion to the yield reduction observed. Seed treatment with Ceresan for a three-year period was shown to have no appreciable effect in controlling this disease. The fact that the disease is seed-borne is well illustrated by successive plantings in paired plots, one plot yielding low, the other yield-

[1]Contribution from Department of Agronomy and Soils, Montana State College, Agricultural Experiment Station, Paper No. 299 Journal Series.

[2]Associate Agronomist, Montana Agricultural Experiment Station.

Table 1. Comparison of yields of Glacier barley grown from non-treated seed from "disease-free" and "diseased" seed sources, at Bozeman, Montana, for the five-year period 1947-51.

Year	Yield (bu./A) from "Disease-free" seed	Yield (bu./A) from "Diseased" seed	Decrease in yield associated with "diseased" seed: Bu/A	Decrease in yield associated with "diseased" seed: Percent
1947	99.6[a]	68.7	30.9	31
1948	100.8	81.2	19.6	19
1949	116.0	68.1	47.9	41
1950	75.4	56.3	19.1	25
1951	81.0	50.0	31.0	38
Average	94.6	64.9	29.7	31

[a] Theoretical yield (see text)

Table 2. Yield of Glacier barley grown at Bozeman, Montana, from seed from "disease-free" and "diseased" sources, treated and not treated, for the three-year period, 1949-51.

Year	Source	Seed treatment: None Yield bu/A	Seed treatment: Ceresan[a] Yield bu/A	Difference[b] measured in: Bu/A	Difference[b] measured in: Percent
1949	"Disease-free"	116.0	120.0	+ 4.0	+ 3
1950	"Disease-free"	75.4	78.3	+ 2.9	+ 4
1951	"Disease-free"	81.0	77.8	- 3.2	- 4
Average	"Disease-free"	90.8	92.0	+ 1.2	+ 1
1949	"Diseased"	68.1	84.6	+16.5	+24
1950	"Diseased"	56.3	56.7	+ 0.4	+ 1
1951	"Diseased"	50.0	45.1	- 4.9	-10
Average	"Diseased"	58.1	62.1	+ 4.0	+ 7

[a] New Improved Ceresan used in 1949, Ceresan M used in 1950 and 1951.
[b] Due to seed treatment.

ing high.

The presence of this disease in the seed of Montana's recommended varieties constitutes a serious threat to the State's 18 million bushel barley crop.

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MONTANA AGRICULTURAL EXPERIMENT STATION

NEW EVIDENCE ON VIRUS DISEASES IN BARLEY

H. H. McKinney

Eslick (3), for the past five years has observed that certain collections of Glacier barley grown in Montana show reduced yields, particularly when grown from seed from certain low yielding plots. The plants in the affected plots were slightly stunted, and sometimes expressed chlorotic markings on the foliage, suggesting a virus disease, possibly barley stripe mosaic (false stripe), the virus of which is known to be seed borne (6, 7). This situation was brought to the attention of the writer by G. A. Wiebe who obtained seed of Glacier barley from R. F. Eslick. Seed was supplied from six high yielding sources and from six low yielding sources.

EXPERIMENTAL RESULTS

Comparisons were made in the greenhouse between the 12 sources of Glacier barley seed, and virus-infected seed of the following ten barleys: Atsel C.I. 6250, Brandon, Chevalier C.I. 156, Chevalier C.I. 200, Chevron C.I. 1111, French Chevalier C.I. 175, Kentucky No. 1 C.I. 6050 (winter), Oderbrucker C.I. 4666, Wong C.I. 6728 (winter), and an unidentified purple seeded hulless spring type. The infected seeds of these barleys, with the exception of Atsel, were from diseased plants that had been inoculated with the type culture of the barley stripe mosaic (false stripe) virus. In the case of Atsel, the infected seeds were from diseased plants that had been inoculated with the barley mosaic virus reported by Wadsworth (10), and which the writer now regards as barley stripe mosaic (false stripe) virus. About 40 seeds of each barley were sown November 26, 1952 in steamed soil contained in 6-inch clay pots. The greenhouse temperature was near 64° F. during the night and on overcast days, and rose to 70° to 75° on bright days.

The first signs of chlorotic markings appeared the second week after seeding in the ten Beltsville varieties, and two weeks later in the Glacier variety. Symptoms developed rapidly and strikingly in most of the diseased plants in the Beltsville varieties, whereas those in the Glacier variety were slow and mostly too weak for positive diagnosis. By January 9, nonparasitic brown spotting had developed to such a degree in the Glacier plants that weak chlorotic markings were probably obscured in some leaves. At this time, unmistakable signs of stripe mosaic were evident in from 39 percent to 78 percent of the plants in eight of the Beltsville varieties, and in 20 percent to 30 percent of the plants in the two others, whereas, Glacier expressed chlorotic markings that were mostly weak, in only 22 percent of the plants in the six low yielding sources, and in 3.3 percent of the plants in the six high yielding sources. Although the nonparasitic brown spotting made it impossible to diagnose stripe mosaic markings accurately in most of the Glacier plants after January 9, it was reasonably evident that most of the infected plants failed to develop even moderate signs of stripe mosaic after that date.

The weakness of the symptoms in Glacier necessitated assays for virus in each plant, but lack of time and space limited this detail to 37 plants each of one high yielding source, No. 22, and one low yielding source, No. 18. These assays revealed the presence of virus in only two plants from the high yielding source, neither of which had expressed detectable symptoms prior to the assay. Virus was detected in 33 plants from the low yielding source, 17 of which had very mild to moderate signs of chlorotic stripes and mottling. Two of these plants expressed the brown stripes that earlier observers accepted as the criterion for false stripe disease, then regarded as being nonparasitic.

Inoculations made into Chevron barley, Michigan Amber wheat, and Golden Giant sweet corn with virus isolates from Glacier suggest that they are mostly mild-symptom inducing strains of the stripe mosaic (false stripe) virus. However, positive identifications will depend upon future studies of the properties of the isolates.

Pot tests (Table 1) show that barley yields are reduced appreciably by stripe mosaic resulting from manual inoculations with virus of the type culture, and by the carborundum leaf wiping method (5). Reductions in yield tend to be correlated with the severity of the disease. These data are from tests of 18 healthy and inoculated plants of each variety grown in pots set out of doors. The symptoms in the Oderbrucker variety were very mild, and resembled those observed in some of the diseased plants of the Glacier variety. In manual inoculation tests, the type culture of the virus has induced severe symptoms in Glacier.

Several of the earlier collections of the stripe mosaic virus give evidence of strains. The isolates made directly from barley have infected only occasional oat plants, but isolates from these in turn have infected most of the oat plants inoculated. Also, there is some seed trans-

Table 1. Reduction in yield and seed size in five barleys inoculated with the stripe mosaic (false stripe) type virus in the seedling stage. Seeded May 2, 1951.

Varieties	Healthy plants		Diseased plants				
	Total wt. of seed	Kernel weight[a]	Total wt. of seed	Kernel weight[a]	Reduction in yield	Delay in heading	Disease ratings[b]
	gm	mg	gm	mg	%	days	
Brandon	45.62	34.4	22.72	32.6	50.20	4	3
Chevalier C.I. 156	51.10	37.0	39.30	34.3	33.10	2	2
Chevalier 2 C.I. 200	44.22	47.7	24.32	31.2	45.00	11	4
Chevron C.I. 1111	44.88	26.9	4.36	19.6	90.29	13	5
Oderbrucker C.I. 4666	34.78	34.4	20.78	33.2	40.25	2	1

[a] Based on 100-kernel samples
[b] Disease ratings: 1=Very mild, 2=Mild, 3=Moderate, 4=Severe, 5=Very severe.

mission of the oat strain in oats. What appear to be mild or semi-mild strains of the virus have been isolated from Union Winter barley C.I. 583, collected in the nursery of G. W. Bruehl at Brookings, South Dakota, and from a hybrid barley (Feebar x Trebi) from the nursery of Orrin J. Webster, Lincoln, Nebraska. Furthermore this hybrid expressed weak stripe mosaic in 54 out of 56 plants when grown in the greenhouse with the Glacier plants.

Tests conducted in the greenhouse indicate that the spray method of inoculation developed for use in the field with the wheat streak mosaic virus (8) is satisfactory for use with the barley stripe mosaic virus. This will facilitate testing barleys in the disease-resistance breeding program, and the production of large stocks of infected seed for experimental purposes.

BROME GRASS MOSAIC VIRUS IN BARLEY

What appears to be the brome grass mosaic virus (4, 9) or a strain of it, was isolated from leaves of the barley hybrid (Feebar x Trebi) collected at Lincoln, Nebraska. So far as known, this virus is not seed borne. Attempts to isolate it from barley seedlings grown from the seed of the hybrid have failed. However, some of these seedlings expressed mild and severe stripe mosaic, and virus was isolated. Although the viruses of barley stripe mosaic and of brome grass mosaic show certain similarities, appreciable differences exist. This is the first time a virus resembling that of brome grass mosaic has appeared in any of the hundreds of isolations made at Beltsville from field-grown small grains collected from many different areas.

BIOLOGICAL CHARACTERISTICS OF THE TYPE CULTURES OF BARLEY STRIPE MOSAIC VIRUS AND BROME GRASS MOSAIC VIRUS

The barley stripe mosaic virus and the brome grass mosaic virus transmit very easily by the carborundum leaf-wiping method described for other viruses of the small grains (5). They can be worked with over a relatively wide range of culture temperatures from 60° F or below to the summer temperatures obtaining at Beltsville, Maryland. However, with the small grains and other cool-temperature plants, strong expressions of symptoms are favored when the temperatures are cool[1]. The incubation periods are about three days at warm, and longer at cool temperatures.

The BARLEY STRIPE MOSAIC (false stripe) VIRUS was isolated from a diseased plant of Chevron barley that became infected via the seed. The culture apparently is either a mixed population of two or more mutant strains that are essentially compatible in Chevron barley, or is largely one type, the mutants of which possess low degrees of compatibility with it. When inoculation is by the carborundum leaf-wiping method, wheat and barley plants rarely fail to become infected. The local and early systemic infection usually causes rather acute chlorosis in

[1] In line 6, p. 564, of the abstract on the barley virus (7), 74°C should read 24°C.

Chevron and certain other barleys, and particularly in Michigan Amber wheat. In wheat, this chlorotic tissue sometimes is white. Chlorosis tends to become less severe as the plants develop, especially in wheat. In Chevron and other varieties of barley studied, the chlorotic markings range from a few to many, narrow to wide, short to long, continuous or broken stripes, and sometimes large mottled chlorotic areas. The chlorotic colorations may be light green, yellow, cream, gray, or bleached white. In barley and wheat, this virus has shown a greater tendency than any other to destroy the carotinoid pigments well in advance of death of the chlorotic tissue. This is the only virus that has induced the brown stripes[2] (false stripe) in barley in these studies. The virus infects a high percentage of the seedlings of Golden Giant sweet corn, inducing local signs, yellow to bleached stripes and wide bands in the leaves, with marked stunting and leaf malformations when systemic invasion is rapid, rarely killing seedlings; it has induced chlorotic and brown markings in all inoculated seedlings of smooth crabgrass (Digitaria ischaemum), in 3 out of 40 inoculated seedlings of smooth brome grass (Bromus inermis), chlorotic markings only in 4 out of 167 oat plants, and local lesions in the Samsun variety of tobacco. In smooth crabgrass the infection seems to be confined largely if not entirely to the stalks receiving the inoculation. There is no evidence of infection in Scotia beans or Early White Spine cucumbers. The virus is seed borne in all of the varieties of barley studied, in Michigan Amber wheat and in oats (an oat strain), but apparently not in Golden Giant sweet corn.

Using infected wheat leaves as source material, the dilution-end-point of the virus in expressed juice and distilled water was slightly beyond 10,000 x, and the thermal-death-point was near 68° C (154.4° F) for 10 minutes. Traces of the virus have passed the Berkefeld "N" filter, pore size averaging 5 to 7 microns. The inactivation-time in air-dried clipped leaf tissue at room temperature was 35 to 40 days, but when the leaves were left on the infected plants the death of the virus followed closely the death of the leaves. The virus seems not to overseason in the soil.

The BROME GRASS MOSAIC VIRUS type culture (4, 9) induces light green to yellow streaking and/or mosaic mottling in the leaves of smooth brome grass, barley, oats, wheat, and many other grass species in eight or more tribes. Almost total destruction of the carotinoid pigments in the chlorotic tissue sometimes occurs in Michigan Amber wheat, leaving the chlorotic tissue almost white. In the small grains studied, the symptoms sometimes become very faint, or they may disappear. Brown markings sometimes appear in the leaves of infected smooth crabgrass, but thus far, they have not been observed in barley known to be infected only with this virus. The virus induces local necrotic lesions and systemic necrosis which is lethal to all inoculated seedlings, and to a low percentage of the older and more resistant plants of Golden Giant sweet corn; it induces local infection and usually lesions in Samsun tobacco, Scotia beans, Early White Spine cucumbers, garden beets, sugar beets (U. S. 215 x 216), and Swiss chard; it induces local chlorotic spots in the inoculated leaves of all the plants, and systemic chlorotic spots in leaves of some of the plants of lambsquarters.

Using infected smooth brome grass leaves for the source material, the dilution-end-point of the virus in distilled water was between 100,000 and 300,000 x, and the thermal-death-point did not exceed 79° C (174.2°F) for 10 minutes. This point was incorrectly given as 80° C in an earlier report (4). The virus passed a fritted glass filter having an average pore size of 0.780 microns. The inactivation-time of the virus in air-dried clipped leaf tissue at room temperature was beyond 12 months, but when the leaves were left on infected plants, death of the virus seemed to follow closely death of the leaves. So far as known, the virus is neither seed borne nor soil borne.

DISCUSSION

The presence of seed borne virus in about 90 percent of the plants grown from seed from a low yielding plot of Glacier barley, despite the fact that most of the infected plants showed few

[2]Several types of nonparasitic leaf spots in barley have been illustrated by Christensen (1) and Dickson (2). Dickson's illustration, figure 4 B, page 20, corresponds to the brown-stripe phase (false stripe) of stripe mosaic, suggesting that the legends for this figure and for figure 4 A are reversed. In the present studies some of the nonparasitic spots were chlorotic and others were dark brown. They started as flecks or as small irregular brown spots, these latter sometimes increasing in size to as much as 3 mm x 4 mm in Glacier barley. When numerous, these various spots can mask the expressions of stripe mosaic. Attempts to link the aforesaid chlorotic and brown spots with the virus of stripe mosaic (false stripe) or with any other virus have met with failure.

or no virus disease symptoms, provides an explanation for the "running out" of certain varieties of barley as claimed by many farmers. No sound genetic explanation for such "running out" has been advanced.

Similar claims for "running out" have been made for other crop plants, particularly potatoes and sugarcane. Accumulated knowledge concerning virus diseases of these vegetatively propagated crops has provided an explanation for their gradual deterioration in yielding capacity under certain circumstances. There are many mild latent virus diseases that can be detected only by close observation and through suitable transmission studies.

Tests should be initiated to determine the extent to which seed borne virus is present in barley seed stocks. The plants should be examined closely for any light green or bleached streaks or mottling in the leaves, as these markings are much more general than the brown stripes. Leaf samples from doubtful plants should be assayed for the presence of virus. In this connection, studies are necessary to determine if the present method of sampling is trustworthy for detecting all cases of infection. On the other hand, the need for these assays will be eliminated or greatly reduced if it is found that the environmental conditions can be selected or adjusted so as to bring stripe mosaic to expression in all or nearly all of the infected plants in a test sample. Until methods for killing the virus in the seed may be devised, the indexing and selecting of disease-free stocks should provide protection against extensive distribution of heavily infected seed stocks.

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REACTION TO STEM AND LEAF RUST AND A SOIL BORNE VIRUS OF HYBRID SELECTIONS OF WHEAT X AGROPYRON X WHEAT AND WHEAT X WHEAT

W. J. Sando

The high resistance of certain Agropyron species to some of the important wheat diseases has stimulated interest in the breeding of disease-resistant wheats by crossing with Agropyron.

In the fall of 1951 seed from 79 selections of wheat x Agropyron x wheat, and wheat x wheat hybrids, were distributed from Beltsville, Maryland, to nine other stations in different sections of the United States and Mexico and to St. Croix in the Virgin Islands. The reaction of these selections to leaf and stem rust (Puccinia rubigo-vera var. tritici, P. graminis var. tritici) and viruses at each reporting station is recorded here. The writer made the crosses and selections and recorded the rust notes at Beltsville. Cooperators who supplied the data from the other stations listed in Table 1 are: F. L. Patterson, Lafayette, Indiana; A. M. Schlehuber, Stillwater, Oklahoma; R. W. Earhart, Gainesville, Florida; C. O. Johnston, J. W. Schmidt, and E. C. Heyne, Manhattan, Kansas; G. K. Middleton and J. G. Moseman, Raleigh, North Carolina; R. G. Shands, Madison, Wisconsin; I. M. Atkins, Denton, Texas; and N. E. Borlaug, Mexico City, Mexico. The rust data from the Virgin Islands were recorded by C. V. Lowther. Seed also was sent to F. C. Elliott at Pullman, Washington, where it was inoculated with bunt races T-13 and T-16, but the experiment failed there because of severe winterkilling.

Insufficient seed of certain selections precluded their distribution to all stations, which accounts for certain omissions seen in Table 1. Although several systems were used to record the data, the various selections can be evaluated readily with respect to their rust and virus resistance. Selections 16, 28, 29, 30, 36, 59, 73, 74, 82, 98, and 99 (Table 1) show outstanding resistance to stem and leaf rust at most of the stations where they were grown.

Most of these rust-resistant selections have speltoid-like spikes and other undesirable Agropyron characters that probably may be eliminated by further hybridization with wheat. Of the 79 selections tested, 71 were from hybrids of wheat x Agropyron elongatum x wheat, 1 was from wheat x A. intermedium x wheat, 5 were from interspecific hybrids of wheat, and 2 were natural hybrids of bread wheats. The parentage of the 79 selections is shown in Table 2.

At Gainesville, Florida, the Purplestraw variety used as a check was completely susceptible to leaf rust. The report from Lafayette, Indiana, omitted any data on susceptible checks but some of the selections tested showed a field reaction to leaf rust of 100 percent. Of the two varieties used as checks at Denton, Texas, Tenmarq had 30 percent leaf rust and Denton had 15 percent. Practically no stem rust developed in the nursery at Denton. Pawnee was used as a check variety at Stillwater, Oklahoma, and was reported as susceptible to leaf rust, but no stem rust developed there. Minhardi, the check variety at Madison, Wisconsin, had 20 to 30 percent leaf rust and 20 to 40 percent stem rust. Winter survival at Madison was poor due to low temperatures. Maximum leaf rust infection in the field was not obtained on the Pawnee check variety at Manhattan, Kansas, because of unfavorable weather conditions. Spreader rows of wheat at Manhattan were inoculated with a leaf rust composite of races 5, 9, 15, 105, 126, and several others, and with a stem rust composite of races 17, 38, and 56. In addition to the field observations on stem rust, results from inoculations with 15B stem rust of seedling wheat plants grown in a greenhouse at Manhattan are included in Table 1. The Purplestraw check variety at Beltsville, Maryland, showed a stem and leaf rust infection of 80 to 90 percent. The plants at St. Croix, Virgin Islands, were inoculated with a composite of biotypes of race 15B obtained from Dr. E. C. Stakman. The rust notes reported in Table 1 are from plants that had not advanced beyond the rosette stage. The Lee check variety developed 60 to 80 percent rust.

At Obregon, Mexico, the Mentana check variety showed a stem rust infection of 85 percent although the selections tested showed 0 to 100 percent infection. The leaf rust notes from Obregon were incomplete and not included in Table 1. The Mentana check variety at Chapingo, Mexico, showed a stem rust infection of 80 percent in the seedling stage and 40 to 60 percent in the adult plants. The leaf rust readings at Chapingo showed that more than one-half of the selections had good resistance.

At Raleigh, North Carolina, only 35 of the 79 selections yielded reliable data, but 20 of them appeared to be homozygous for resistance to a soil-borne virus. Twleve of the selections segregated for resistant and susceptible plants and three were homozygous for susceptibility.

It is noteworthy that Nos. 59, 98, and 99, which were resistant to a soil-borne virus in North Carolina, also were extremely resistant to leaf and stem rust at most of the other sta-

Table 1- Reaction of wheat x Agropyron x wheat and wheat x wheat segregates to leaf and stem rust, and a soil borne virus.

1951 Progeny No.	Location, Severity, and Response: Leaf Rust: Gainesville, Florida		Lafayette, Indiana		Denton, Texas		Madison, Wisconsin		Stillwater, Oklahoma		Manhattan, Kansas		Beltsville, Maryland		Chapingo, Mexico		Stem Rust: Madison, Wisconsin		St. Croix, V. I.		Chapingo, Mexico		Obregon, Mexico		Manhattan, Kansas		Beltsville, Maryland		Soil virus: Raleigh, N. C.
	Adult	%	Adult	%	Adult	%	Adult	%	Adult		Adult		Adult		Adult		Adult	%	Adult	%	Seed-ling	%	Adult	%	Adult		Seed-ling 1/		Adult	
2	T	0	T	-		S	R	T-L	S	25	80	40	50	100	S	3	M-H	-											
3	T-10	0	15	-		S	R	T-L	S	15	80	0	90	100	S	3	M-H	-											
5	T-10	0	10	1		S	R	T-L	-	15	90	0	T	40	MS	1	O-T	-											
6	T-50	0				S	R	O-T	-			40	90	40	S	2	M-H	-											
7	T-20	0	T	-		S	R	O-T	R	-	0	40	0	0	R	1	0	S											
8	T-30	0	T	-		S	R	O-T	S	-	80	40	40	40	MS	1	O-T	-											
10	T	0	T	-		S	S	T-L	S	-	25	40	90	100	MS	4	M-H	R-S											
11	T	0	T	-		-	R	0	R	25	T	T	80	35	S	1	M-H	-											
13	40-50	0	T	0		S	S	T-L	-	O-T	40	40	80	85	MS	4	M-H	-											
16	T	0	T	0		S	R	0	R	0		0	0	0	Seg.	0	O-T	-											
17	T	0	T	0		R&S	R	O-T	R	1-60	90	0-30	30	75	S	3	O-T	-											
18	T-40	0	10	T		R	Seg.	O-L	R	5	60	0-30	20	65	S	2	L-M	-											
19	0					R	R	0	R			0-30	40	75	MS	2		-											
20	60	0	10	5		S	S	L	R	T-60	80	O-T	40	-	S	1-4	M	-											
21	40-70	0	15	-		S	S	M	-	-	90	90	80	100	MS	4	M	-											
22	T-60	0	5	T-10		S	S	M		5-20	30	40	90	100	MS	4	M	R-S											
23	T-70	100	20	20		S	S	H	-	40	60	80	90	100	MR	4	M-H	R-S											
24	20-60	100	20	0		R	Seg.	L-H	S	0-10	30	60	80	100	Seg.	3	M-H	-											
27	0	0	T			R	R	0	-	-	10	0	5	100	R	0;	0	-											
28	T	0	5	Seg.		S	Seg.	0	R	0	40	40	0	0	R	1-2	O-L	-											
29	T	0				S	R	0	R			O-T	0	0	R	0;	0	-											
30	T	0	T	0		R	R	0	R	0	0	40	0	0	MR	1	0	-											
31	T-60	T	T	0		S	R	0	R	0	0	O-T	0	0-40	R	1	0	-											
32	20-40	0	25	-		S	Seg.	-	S	-	30	30	30	0-100	Seg.	1-2	O-T	-											
33	T	0	T	0		R	R	-	R	0	0	30	0	0-100	MR	0;	T-H	-											
36	T	0	T	0		-	R	0	R	0-5	0	O-T	0	0	MR	0;	L-M	-											
37	T	0	5	-		R	R	0	S	-	60	20	80	100	S	2	0	-											
38	T-20	0	T	T-10		R	Seg.	0	R	10-30	90	20	15	100	MS	2	T-L	-											
39	T-60	0	T	T-20		S	Seg.	H	S	20	90	O-T	40	-	MS	1-4	H	-											
40	50	0				S	Seg.	L	MR-S			O-T	0-20	0	R	4	O-M	-											
41	70	50	10	3		S	S	M-H	S	10	40	0-40	5	100	Seg.	1-4	O-T	-											
42	30-80	50				S	S	T-H	S			40	50	-	MS	2-4	H	-											
43	T	0	5	-		R	R	O-T	R	-	40	-	50	-	MS	0;	O-T	-											
44	20	0				R	R	O-T	MR			-	5	50	S	3+	O-H	-											
45	T	-	T	-		R	R	0	MR	-		-	10	O-T	S	2	T-L	-											
46	T-50	-				R	S	0	0			O-T	0-20	-	S	1-3	0	-											
48	0-40	-				S	R	O-T	-	-		20	50	-	MS	2	L-M	-											
49	T-50	T-50	15	-		S	Seg.	O-T	S	-	60	30	40	85	Seg.	2+	O-T	R											
51	10-50	100	35	3		S	Seg.	M	MR	0-10	40	20	40	75	Seg.	0;-3	T-L	R											
52	T	0	20	T-30		R	R	O-M	R	T-60	40	O-T	30	-	MR	2-4	T-L	-											
53	T	0	5	T-10		R	R	O-M	MR	30		0-10	30	90	MS	2	M	S											
54	30-70	100	T	30		S	S	T-H	MR	T-40	80	30	40	100	Seg.	3	M-H	R											
55	T	100				S	R	O-T	-	-		30	0-40	100	R	0;-2	0	-											
56	T-80	0	T	T		R	R	O-H	S	5	90	0-40	40	20-85	Seg.	3-4	O-H	-											
58	T-20	0-100	T	0		S	Seg.	O-H	R	0		0-10	0	75	Seg.	1	O-H	-											
59	40	0-100	5	T		S	R	O-T	R	0	0	O-T	0	0	R	1-	0	R											
60	T	50	20	T		S	Seg.	L-M	R	T+	60	0-30	0	85	R	1	0	R											
61	20	T	5	5		S	R	L	R	-	60	0-30	5	0	MR	1	M	S											
62	30	T	5	0		S	R	O-T	R	0	0	0-20	0	0-10	R	1-	0	-											
63	T	0				S	R	L	R			O-T	0	65	R	1-	0	-											
65	0-10	0	5	T-5		S	R	O-T	MR	20	20	0-30	40	75	MR	1	T-L	R-S											
66	T-30	0	T	20		S	R	T-L	S	30	10	40	60	100	MR	2+	M	R											
68	T-40	T-100	10	1		S	Seg.	T-L	-	T-20	40	0-30	10	5-75	Seg.	1-3	O-M	R											
69	T	T	T	-		S	R	O-T	-	-	90	0-30	15	75	R	1-4	0	R											
70	T-10	T	T	T-5		S	R	M-H	S	30		30	40	75	Seg.	3	M-H	R											
71	T-50	T	T	T		R	R	O-T	-	0-10	5	0-30	15	-	R	1+	O-T	R-S											
72	T-50	0	20	T		S	R	O-T	-	T-10	80	0-40	10	100	Seg.	1-3	0	R											
73	T	-	T	-		R	R	0	S	-	20	O-T	0	T	R	0-1	0	-											
74	T	-	T	-		R	Seg.	O-T	R	-	20	O-T	0	T	S	0-1-	T	-											
75	0-80	100	20	25		S	S	O-T	S	30	60	40	50	40	S	1-4	H	R-S											
76	T-40	0-100	5	15		S	Seg.	T-H	S	20	60	40	0	70	Seg.	1-3	H	R-S											
77	T-20	0	5	20		S	R	O-L	MS	20 -50	75	40	30	40	Seg.	2+	L-	-											
78	10-60	0	T	-		S	R	T-H	R	-	20	30	0	0-100	R	1-4	T-M	-											
79	T-20	0	10	T-20		R	R	O-L	R	10-50	60	30	60	50	MS	2+	O-L	R-S											
80	0-20	0	5	T-5		S	Seg.	O-M	R	10-40	40	30	30	-	R	1-4	M	R-S											
81	60	100	5	25		S	Seg.	H	S	50	80	40	90	100	S	4	M-H	R-S											
82	T	-	T	-		R	R	O-T	-	-		O-T	0	35	MS	0-1-	0	-											
84	0	T	T	T-5		S	Seg.	O-T	S	20-40	45	30	90	-	S	2+	T-L	R-S											
86	T	100	T	T-		S	R	-	-	T-5	80	10	20	-	R	1	-	R											
88	80	50	15	40		S	S	M	-	40	90	60	100	100	S	4	M	R											
89	T-50	0	T	0-40		S	R	O-T	R-S	0-30	80	0-40	0-100	0-100	R	1-4	O-H	R											
90	20-60	0	10	T-10		S	R	L-H	-	0-40	80	30	60	40-100	MS	1 2-4	O-T	R											
91	T	100	5	0		S	R	O-T	R	0	0	10	0	-	R	0;	0	R											
94	60	0-100	20	T-20		S	Seg.	L-M	S	0-40	45	20	15	100	Seg.	1-3	O-L	R											
95	40-60	-	10	T-50		S	Seg.	M-H	S	0-50	0	20	100	-	MS	0; 1-3	H	R-S											
96	T	0	10	T		S	R	L	S	3		O-T	0	40	Seg.	1-	0	R											
98	T-20	0	5	O-T		S	R	0	S	0	0	10	0	-	R	1-	0	R											
99	T	100	T	0		S	R	0	R	0		O-T	0	0	R	1-	0	R											
100	40-80	50	T	30		S	Seg.	L	R	50	35	60	0?	30-100	Seg.	3-4	M	R											
Check	60-70		30	20-30		S		80-90	S	20-40	60-80	80	40-60	85		80-90	80-90												

Legend: 0 = disease free, T = trace, L = light infection, M = medium infection, H = heavy infection, R = resistant, MR = moderately resistant, MS = moderately susceptible, S = susceptible, Seg. = segregating, - = stand insufficient or plant development unfavorable for procuring data.

1/ 0-4 No evidence of rust infection to completely susceptible.

Table 2. Parentage of selections of wheat x Agropyron x wheat and wheat x wheat, hybrids distributed to cooperators for disease evaluation.

1951 Progeny No.	Parents
2	Chinese x A. elongatum x Harvest Queen and Purplestraw
3	"
5	Chinese x A. elongatum x Arlando and Leapland x Comet 125
6	Chinese x A. elongatum x Federation and Kinney x Prelude x Arlando x Pilot
7	Chinese x rye x Chinese x A. elongatum x Rising Sun x Purplestraw and Leapland
8	Hussar x Trumbull x Webster x Purplestraw x Chinese x rye x Chinese x A. elongatum x Forward
10	"
11	Sol x A. elongatum x Leapland
13	Redhart 5 x Chinese x A. elongatum x Federation and Kinney x Prelude
16	Chinese x A. elongatum x Purplestraw C.I. 1915 x Red Rock x Comet x Red Rock
17	Chinese x A. elongatum x Federation and Kinney x Prelude x Forward
18	Chinese x rye x Purplestraw x A. elongatum x Leapland and Arlando x Baart
19	Chinese x A. elongatum x Federation and Kinney x Prelude x Red Rock
20	Sol x A. elongatum x Leapland
21	Chinese x A. elongatum x Arlando and Leapland
22	Hussar x Trumbull x Webster x Purplestraw x Chinese x rye x Chinese x A. elongatum x Forward
23	Trumbull x Caldwell 256 x Khapli emmer
24	Chinese x A. elongatum x Leapland x Arlando
27	Chinese x A. elongatum x Federation and Kinney x Prelude x Yorkwin
28	Chinese x rye x Chinese x A. elongatum x Rising Sun x Purplestraw and Leapland
29	T. vulgare x A. elongatum
30	Chinese x rye x Chinese x A. elongatum x Forward
31	"
32	Chinese x rye x Chinese x A. elongatum x Rising Sun x Purplestraw and Leapland
33	Chinese x rye x Chinese x A. elongatum x Forward
36	"
37	Chinese x A. elongatum x Federation and Kinney x Prelude x Forward
38	"
39	Sol x A. elongatum x Leapland
40	Chinese x A. elongatum x Arlando and Leapland x Comet 125
41	Chinese x rye x Chinese x A. elongatum x Lutescens and Hostianum
42	Chinese x rye x Chinese x A. elongatum x Federation and Kinney x Prelude x Redhart 5
43	Rising Sun x A. elongatum x Illini Chief x Purplestraw x Premier
44	T. vulgare x Agropyron, Suneson 464-3-1 (46)
45	" 700-P-14 (46)
46	" 118C-1 fr. 198D2
48	Chinese x A. elongatum x Federation and Kinney x Prelude x Nebred
49	" " x Red Rock
51	Chinese x A. elongatum x Harvest Queen and Purplestraw
52	Chinese x A. elongatum x Comet x Nebred
53	Federation Sacaton 78 (38) x Sol x A. elongatum x Leapland
54	T. vulg. x A. elongatum [11B O.B. 1 (46)]
55	Chinese x A. elongatum x Federation and Kinney x Prelude x Arlando x Pilot
56	Arlando x T. timopheevi x Hope x Baart
58	Chinese x rye x Chinese x A. elongatum x Rising Sun x Purplestraw and Leapland
59	Chinese x A. elongatum x Harvest Queen and Purplestraw
60	T. vulgare volunteer in field.
61	Ta. #2 (44) Stem Rust Resistant T. vulgare
62	Chinese x rye x Chinese x A. elongatum x Rising Sun x Purplestraw and Leapland

Table 2 (continued)

1951 Progeny No.	Parents
63	Chinese x A. elongatum x Arlando and Leapland x Comet
65	Federation Ab 6 (38) B x Sol x A. elongatum x Leapland
66	Chinese x A. elongatum x Comet x Red Rock
68	Arlando x T. timopheevi x Hope x Baart
69	Fulhio x Yaroslav emmer
70	Chinese x rye x Purplestraw x A. elongatum x Leapland and Arlando
71	Chinese x A. elongatum x Federation and Kinney x Prelude
72	Hussar x Trumbull x Webster x Purplestraw x Chinese x rye x Chinese x A. elongatum x Forward
73	Rising Sun x A. elongatum x Illini Chief x Purplestraw x Premier
74	"
75	"
76	Chinese x A. elongatum x Federation and Kinney x Prelude x Nebred
77	Rising Sun x A. elongatum x Illini Chief x Purplestraw x Chinese x A. elongatum x Federation and Kinney x Prelude x Carala
78	T. sphaerococcum x A. intermedium x Yorkwin LEP 113 (47)
79	T. vulgare x A. elongatum, Suneson 700-P-14 x Trumbull x W 28-Fultz Sel.-Hungarian x Trumbull-Frondoso, Y2375 (46)
80	"
81	T. vulgare Sac 75 (38) x Sol x A. elongatum x Leapland and Purplestraw
82	Rising Sun x A. elongatum x Illini Chief x Purplestraw x Premier
84	Chinese x A. elongatum x Federation and Kinney x Prelude x Red Rock
86	T. vulgare x A. elongatum Wa 109-4-14-6
88	Chinese x A. elongatum x Harvest Queen x Purplestraw
89	Chinese x A. elongatum x Arlando x Leapland x Comet 125
90	Chinese x A. elongatum x Federation and Kinney x Prelude x Chinese x A. elongatum x Federation and Kinney x Prelude
91	Chinese x rye x Chinese x A. elongatum x Rising Sun x Purplestraw and Leapland
94	Arlando x T. timopheevi x Forward x Hope x Baart
95	Chinese x rye x Chinese x A. elongatum x Forward x Arlando
96	Chinese x A. elongatum x Arlando and Leapland x Comet 125
98	Chinese x rye x Chinese x A. elongatum x Rising Sun x Purplestraw and Leapland
99	Chinese x A. elongatum x Arlando and Leapland x Comet 125
100	Hussar x Trumbull x Webster x Purplestraw x Chinese x rye x Chinese x A. elongatum x Forward

tions where they were grown.

DIVISION OF CEREAL CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, DEPARTMENT OF AGRICULTURE

REACTIONS AT BELTSVILLE, MARYLAND, OF SEGREGATES FROM HYBRIDS OF WHEAT X AGROPYRON ELONGATUM X WHEAT TO EIGHT RACES OF PUCCINIA GRAMINIS TRITICI

W. J. Sando and C. V. Lowther

The superior rust resistance of certain Agropyron species has long been recognized, but the transfer of this resistance to wheat has presented many difficulties. The rust-resistant types obtained thus far still possess too many of the undesirable Agropyron characters to be

Table 1. Greenhouse seedling reactions of wheat x Agropyron elongatum x wheat hybrids to 8 races of Puccinia graminis tritici.

1951 No.[a]	Pedigree	Reaction[b] to race: 11	15B	17	19	36	38	56	59
3	Chinese x A. elong. x Harvest Queen and Purplestraw	4	4	4	4	4	4	4	4
5	Chinese x A. elong. x Arlando & Leapland x Clark's Comet	0;-1	0	0;-1+	0	0;-1	0	7=0;-1 1=4	0
16	Chinese x A. elong. x T. aestivum R. R. 209 (34) & Comet x Red Rock	0	0;-1	0;-1 1=4	0	1=3+ 5=0;	0	0	0;-1
27	Chinese x A. elong. x Federation & Kinney x Prelude x Yorkwin	0;-1	0;	0;-1	0	0;	0;-1	0;-1	0;
29	T. aestivum x A. elong. x T. aestivum	0	0	0;-1	0	0;	0;	0	0; 1=4
31	Chinese x A. elong. x Chinese x rye x Forward	0	0	0;-1	0;	0;	0;-1	5=0 1=4	0
36	Chinese x A. elong. x Chinese x rye x Forward	0	0	0;	0;	0;	0	0	0
73	Rising Sun x A. elong. x Illini Chief x Purplestraw x Premier	7=0;[c] 2=4	0;-1	0;-1	0;-1	0;-1	4=0;-1 1=4	0;-1	0;-1
74	Rising Sun x A. elong. x Illini Chief x Purplestraw x Premier	0;-1	0;	0;-1	0;-1	0;-2=	0;-1	0;-1	0;-1
96	Chinese x A. elong. x Federation and Kinney x Prelude x Nebred	0;	0;	0;-1‡	0	0;-1+	0	0;-1‡	0
99	Chinese x A. elong. x Arlando & Leapland x Clark's Comet 125	0;	0;-1	0;-1	0	0;-1	0	0	0 1=3

[a] = Sando accession number.
[b] 0-4 = No evidence of rust infection to completely susceptible.
[c] Number of plants and response, respectively.

of value as a grain crop. It appears, however, that with further hybridization a commercial wheat possessing the high rust resistance of the Agropyrons may be possible.

In 1952 11 selections from hybrids of wheat x Agropyron elongatum x wheat made by the senior writer were inoculated in the seedling stage with eight races of stem rust by the junior writer and kept in the greenhouse at a temperature of 70° to 75° F. These selections are fertile and are crossable with wheat. The results of this test are shown in Table 1. All of the selections except Row 3, which is quite susceptible to the eight races of stem rust used, showed a remarkably high resistance to these races. One of the selections, Row 36, possesses higher resistance to the eight races than that known to be present in any wheats thus far tested. Five of the 11 lines were relatively homozygous for reaction to all eight races of rust while the remainder segregated for reaction to one or two races.

Most of the segregates also were highly resistant to powdery mildew (Erysiphe graminis) that developed in the greenhouse and were very resistant to the local races of leaf rust (Puccinia rubigo-vera var. tritici) when grown in the field at Beltsville.

DIVISION OF CEREAL CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, DEPARTMENT OF AGRICULTURE

EFFECT OF STEM RUST ON YIELD, TEST WEIGHT, AND MATURITY OF OATS[1]

P. Rothman and K. J. Frey

A disease which causes major losses in oat yields in the North Central States is stem rust, Puccinia graminis avenae. The extent of damage done is usually controversial and statements relative to the percentage of loss in yield are empirical estimates. It is generally agreed that, if two oat growing areas have equal epiphytotics of stem rust, the greatest loss will occur in the area with the driest atmosphere[2]. Since the major oat growing area in Michigan is located in a humid atmosphere region, stem rust of oats would not be likely to cause yield losses so great as those reported in drier regions. In order to determine the potential damage due to oat stem rust in Michigan, resistant and susceptible oat strains, grown under stem rust epiphytotic conditions, were compared in 1951 and 1952 for yield of grain, weight per given volume of grain (analogous to test weight), and relative maturity as measured by date of heading.

Materials and Methods

The oat strains used in this study were selected from the F_2 progenies of the crosses Wolverine x Hajira-Joanette (A587) and Huron x Hajira-Joanette (A584). The only criterion used in choosing the oat strains was their reactions to races 2 and 8 of stem rust. Only those with identical reaction to both races were suitable, i.e., either resistant, segregating, or susceptible in reaction to both races. Huron and Wolverine are stem rust-susceptible varieties that are well adapted in Michigan while Hajira-Joanette is unadapted and stem rust-resistant.

From 300 F_2 progenies classified for reaction to races 2 and 8 of stem rust, 33 were chosen at random within the various reaction groups. They consisted of six resistant, nine segregating, and six susceptible selections from the A587 cross, and eight resistant and four segregating selections from the A584 cross. These 33 strains plus the three parent varieties were planted in a 6 x 6 triple lattice design with three replications. The plot size was a single row eight feet long with the rows spaced twelve inches apart. Every second row was sown to Huron variety to provide uniform competition for the oat strains and to serve as a rust spreader. The oat strains when yield-tested in 1951 and 1952 were in the F_3 and F_4 generations, respectively.

The planting dates were May 3 and April 23 in 1951 and 1952, respectively. A stem rust epiphytotic was induced by dusting a mixture of talc and spores of races 2 and 8 of stem rust on the Huron border rows when the plants were in the four-leaf stage.

Yields recorded in grams were obtained by harvesting the grain from entire plots and the date of heading was recorded when 50 percent of the heads in a plot had emerged above the flag leaf. For convenience in statistical analysis the heading dates were converted to numbers with one representing June 24, two June 25, etc. Weight per given volume, hereafter referred to as test weight, was taken as weight in grams of the grain in a level full 25 ml. beaker.

Experimental Results

It was assumed in the planning of this study that, if several random selections were made in each of the stem rust reaction groups, resistant, segregating, and susceptible, within an oat cross segregating for this characteristic, the average yielding ability of the three reaction types would be approximately equal. This assumption was based upon the fact that except for the yield genes linked with the stem rust reaction genes the genotypes of selected strains were merely random samples from a common genetic background. Since the resistant parent (Hajira-Joanette) in each cross was inferior to the susceptible parents in yield because it was unadapted to Michigan conditions, any bias caused by linkage should have been in favor of the susceptible group. If the mean yields of the susceptible and segregating groups were lower than that of the resistant group when all three groups were grown under stem rust epiphytotic conditions, the difference could be attributed to the damaging effects of stem rust.

The mean heading dates, test weights, and yields for the various rust reaction groups and the parental varieties are presented in Table 1 while the analyses of variance for these data are presented in Table 2. In the A587 cross in both years, the mean yield of the resistant group was approximately 25 percent higher than that of the susceptible group, a difference which was

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[1]Contributed from the Farm Crops Department of the Michigan Agricultural Experiment Station, East Lansing, Michigan. Journal Article No. 1474.

[2]Greaney, F. J. 1936. Cereal rust losses in Western Canada. Sci. Agr. 16: 608-13.

Table 1. The mean heading dates, test weights, and yields of three oat varieties, and the various stem rust reaction groups in two oat crosses grown in 1951 and 1952.

Cross or variety	Resistance group	Mean date of heading		Mean test weight		Mean yield in grams	
		1951	1952	1951	1952	1951	1952
A584	Resistant	June 30	June 29	40.1	43.0	100.1	90.6
	Segregating	June 30	June 29	37.9	41.5	96.8	82.3
A587	Resistant	July 5	July 2	39.2	39.6	99.8	91.5
	Segregating	July 6	July 2	39.1	39.4	88.1	83.2
	Susceptible	July 9	July 3	36.7	36.8	76.4	71.9
Huron	Susceptible	June 28	June 26	40.3	44.8	86.4	91.3
Wolverine	Susceptible	July 6	June 30	39.7	40.6	93.2	93.0
Hajira-Joanette	Resistant	July 6	July 2	37.0	42.2	72.8	78.3

highly significant. This yield difference can be attributed to stem rust damage. The mean yield of the segregating group was approximately equal to the midpoint between the resistant and susceptible group yields; a fact which would be expected since the ratio of susceptible to resistant plants in the segregating progenies in the F_3 and F_4 was approximately one to one. In both years the segregating group yielded less than the resistant in the A584 cross but neither difference was significant. At first glance at Table 1 it may be disturbing that the yields of the susceptible parents Huron and Wolverine are not different from the mean yields of the stem rust resistant groups. However, it must be remembered that these two varieties have been selected for their high yields under Michigan conditions while the resistant groups were averages of several strains not selected for yield. In fact, in both years one strain in the resistant group yielded about 20 percent higher than either Huron or Wolverine. The stem rust-resistant parent, Hajira-Joanette, yielded less than either of the susceptible parents, which, as stated earlier, indicates that the inferior yields of the susceptible and segregating groups are caused by the stem rust organism. The rank correlation between yield and stem rust severity in 1951 was -.49, a highly significant value. This further demonstrates the association between high yields and low rust readings.

There were significant yield differences among oat strains within the stem rust reaction groups. However, this could be expected since the strains selected within reaction groups probably had different inherent yielding capacities and, even if each were reduced in yield by the same percentage short of complete destruction, there would still be significant differences between strains.

The mean test weight of the susceptible group in the A587 cross was approximately 8 percent less than those of the resistant and segregating groups. This difference was highly significant. The difference in test weight between the stem rust reaction groups in the A584 cross was significant in 1951, but not in 1952. The rank correlation between test weight and stem rust severity in 1951 was -0.50; a highly significant value that shows an association between high test weight and low stem rust severity. The damaging effects of the stem rust disease upon test weight, although not so severe as the losses in yield, were nevertheless considerable and undoubtedly contributed to the reduction in yield.

There is some indication in this study that the heading date of the susceptible oat strains in the A587 cross was delayed by the stem rust disease. In both years the mean date of heading in the resistant group was significantly earlier than that of the susceptible. This delay in plant development could have affected test weight indirectly because lateness is associated with light test weight.

Discussion

The stem rust readings on Huron variety were between 25/100 and 65/100, showing a heavy epiphytotic. Since the infection started in the early stages of plant growth it is surprising that

Table 2. Analyses of variance of data on yield, test weight, and heading date from three oat varieties and thirty-three selections representing different stem rust reaction types from two oat crosses.

Source of variation	Degrees of freedom	Mean squares					
		1951			1952		
		Yield	Test weight	Date of heading	Yield	Test weight	Date of heading
Entries	35	636**	10.7**	52.5**	496**	9.9**	16.9**
Parents vs crosses	1	541	0.0	5.0	85	16.0**	27.0**
Between parents	2	322	9.5	66.5**	193	3.0	29.5**
Between crosses	1	2714**	23.0**	894.0**	685*	46.5**	165.0**
Among selections within crosses	31	592**	10.7**	26.0**	523**	7.5**	11.0**
Among selections in A587 cross	20	867**	9.2**	22.2**	536**	6.2**	6.6**
Between resistance groups in A587	2	2462**	38.5**	92.5**	1738**	21.0**	15.5**
Res vs Sus	1	4923**	64.0**	156.0**	3442**	32.0**	22.0**
(Res + sus) vs Seg	1	0	13.0*	29.0**	35	10.0*	9.0**
Among selections within resistance groups	18	690**	5.9*	14.4**	402**	4.6**	5.6**
Among selections in A584 cross	11	482*	13.5**	32.8**	499**	9.7**	19.0**
Between resistance groups in A584	1	86	39.0**	5.0**	544	5.0	0.0
Among selections within resistance groups	10	522*	11.0**	35.6**	494**	10.2**	20.9**
Error	55	237	3.0[a]	1.7	164	1.5	1.6

[a] 70 d.F.

the yield reduction was not greater than 25 percent, especially in 1952, when the weather was dry during the grand period of growth and the ripening period. It is interesting that the rust pustules were more prevalent on the leaf blades than on the sheaths. This may account for the yield losses being no greater than they were.

Data such as these reported in this paper point out the need for more and continued research in the breeding of rust-resistant oat varieties. Other means of rust control, such as chemical sprays or dusts and barberry eradication, are only partially effective. Chemical controls are uneconomical for a low income crop like oats, and barberry eradication, though effective in preventing early infection and the production of new races through crossing, does not control the wind-borne southern red spores from causing later infection. With rust-resistant oat varieties the grower need not go to any special effort to control the disease because resistance is inherent with the variety. Recently oat growers have had to change varieties often in order to keep ahead of new rust races with the virulence to damage older varieties seriously, but this requires little expense to the farmer.

Summary

In experiments conducted at East Lansing, Michigan, in 1951 and 1952 an epiphytotic of stem rust of oats caused, (a) a 25 percent reduction in yield, (b) an 8 percent reduction in test weight, and (c) a two-day delay in heading of susceptible oat strains where compared with their resistant counterparts from the same cross.

MICHIGAN AGRICULTURAL EXPERIMENT STATION, EAST LANSING

REACTION OF OATS VARIETIES AND SELECTIONS TO POWDERY MILDEW AT LAFAYETTE, INDIANA

John F. Schafer, Ralph M. Caldwell, L. E. Compton, and Fred L. Patterson

Powdery mildew of oats due to Erysiphe graminis avenae Em. Marchal developed in severe proportions in the greenhouse at Lafayette, Indiana, in 1950-51 and 1951-52. This was the first known occurrence of this disease on oats in the greenhouse at Lafayette although powdery mildew of wheat and barley commonly occur. It has been thought that this development may have coincided with the fact that August-planted crops of oats were grown in the greenhouse during the falls of 1950 and 1951, whereas in previous years oats were planted in October or later. However, the recent report by Murphy (2) of a similar occurrence for the first time at Ames, Iowa, also in the fall of 1950 leads one to place more significance on this development. Advantage was taken of the presence of the oats mildew to make a limited test in January, 1951, of the relative development of the disease on 130 varieties and selections of oats. These data are reported in Tables 1 and 2. Disease reactions are given on a scale of "0" to "4+" with "0" indicating no development of mildew, "4+" the greatest susceptibility, and intermediate readings indicating the varying intermediate amounts of mildew development. No "0" readings were obtained, but there was considerable variation in the degree of development of mildew as indicated in the accompanying tables. These data appear partially to corroborate those of Finkner (1) obtained at Ames, Iowa, although the reaction types reported here suggest a higher level of resistance in many lines in this test as compared with the results of the Iowa test.

Table 1. Reaction of spring oats varieties and hybrid selections to powdery mildew in the greenhouse at Lafayette, Indiana, January, 1951.

Name or selection	Reaction
Advance C.I. 3845	2
Ajax C.I. 4157	3-
Algerian P.I. 159207, Botany 50	2-
Amarilla Buck, Botany 36	2
Amarilla La Prevision, Botany 35	1+
Anthony C.I. 2143	1-
Anthony x Bond C.I. 5298	2
Anthony, Bond x Boone C.I. 5218	2
Anthony, Bond x Boone C.I. 5221	2+
Anthony, Bond x Boone C.I. 5224	3
Arkansas 674, Botany 39	4
Beaver C.I. 4521	3
Belar C.I. 2760	1+
Benton C.I. 3910	2+
Berry 333 C.I. 4811	4
Blanca Klein Mar, Botany 37	3+
Bond C.I. 2733	2
Bond x Anthony C.I. 4189	2-
Bond, Double cross A x Nakota C.I. 5330	3+
Bond, Double cross A x Nakota C.I. 5331	2
Bond x Iogold C.I. 4330	3-
Bond x Rainbow C.I. 4253	3+
Bonda C.I. 4329	2-, 4-
Bondvic C.I. 5401	2+
Bonham C.I. 4676	2+

Table 1 (continued)

Name or selection	Reaction
Boone C.I. 3305	2+
Branch C.I. 5013	3-
Cartier	3
Cherokee C.I. 3846	4
Clinton C.I. 3971	3, 4-
Clinton 59 C.I. 4259	3
Clinton 59-1	4+
Clinton x Bond 416-63-2	4+
Clinton x Boone, Cartier C.I. 6641	4-
Clinton x Cartier 419A6-8-1-1	4+
Clinton x Marion C.I. 5441	3-
Clinton x Marion C.I. 5638	3
Clinton x Marion C.I. 5640	2
Clinton x Marion C.I. 5644	2
Clinton x Marion C.I. 5647	3+
Clinton x Marion C.I. 5650	3
Clinton x Marion C.I. 5652	3+
Columbia C.I. 2820	3
Columbia x Clinton C.I. 5629	4-
Craig C.I. 5332	1+
D69 x Bond M3218-2064-4	3+
D69 x Bond C.I. 3841	2
D69, Bond x Cartier 4110A6-2-1-2	4++
D69, Bond x Hajira, Joanette, Victoria 720-15-4-2-1	2
D69, Bond x Hajira, Joanette, Victoria 720-34-1	1+
D69, Bond x Hajira, Joanette, Victoria 720-34-5	1+
D69, Bond x Vanguard 702-5	2+
D69, Bond x Vanguard 702-18	3
D69, Bond x Vanguard 702-18-1-2	3-
Eaton C.I. 3908	1+
Exeter C.I. 4158	4
Glabrota C.I. 2630	1
Golden Beauty C.I. 4810	3+
Golden Rain C.I. 4512	2
Gopher C.I. 2027	1
Gothland C.I. 1898	2
Huron C.I. 3756	4
Green Mountain C.I. 1892	2
Green Russian C.I. 2890	2
Hawkeye C.I. 2484	1+
James C.I. 5015	3
Kent C.I. 3909	2+
Landhafer C.I. 3522	1, 2-
Langgewens PI 159208, Botany 51	1-
Marion C.I. 3247	2-

Table 1 (continued)

Name or selection	Reaction
Markton C.I. 2053	3+
Markton x Rainbow C.I. 3350	3-
Mindo C.I. 4328	3+
Mindo x Landhafer C.I. 5216	3+
Missouri 0-205 C.I. 4988	2
Missouri 0-205 C.I. 5323	4+
Missouri 04201, Columbia x Victoria, Richland C.I. 5297	1-
Moroto x Bond C.I. 3328	2+
Nemaha C.I. 4301	4-
Nemaha x Clinton, Boone, Cartier C.I. 6642	4
Neosho C.I. 4141	2-
Osage C.I. 3991	1-
Rainbow C.I. 2845	2-
Richland C.I. 787	1
Ruakura C.I. 2025	1-
Santa Fe C.I. 4518	1+
Santa Fe x Clinton C.I. 5400	3
Santa Fe x Clinton C.I. 5424	3
Santa Fe x Clinton^2 C.I. 5209	4-
Santa Fe x Clinton^2 C.I. 5210	2
Santa Fe x Clinton^2 C.I. 5211	3+
Santa Fe x Clinton^2 C.I. 5212	3+
Santa Fe x Clinton^2 C.I. 5213	3-
Santa Fe x Clinton^2 C.I. 5214	3-
Shelby C.I. 4372	2-
Sterisel C.I. 2891	1
Sunrise C.I. 982	1-
Tama C.I. 3502	2
Trispernia C.I. 4009	2-
Ukraine C.I. 3259	3+
Vicland C.I. 3611	1+
Victoria C.I. 2401	2+, 3-
White Tartar C.I. 551	2+
Zephyr C.I. 4800	2+

Table 2. Reaction of winter oats varieties and hybrid selections to powdery mildew in the greenhouse at Lafayette, Indiana, January, 1951.

Name or selections	Reaction
Atlantic C.I. 4599	3
Clinton x Forkedeer C.I. 5369	3+
Clinton x Forkedeer 4011-4-31	2+
Clinton x Forkedeer 4011-4-81	1
Clinton x Hairy Culberson C.I. 5368	2+
Clinton x Hairy Culberson, Fulghum Okla. 1460: 406-3-5	1
Colo x Wintok C.I. 5118	1
Coy C.I. 4600	4
Dubois C.I. 6572	4-
Forkedeer C.I. 3170	1+
Forkedeer x Hairy Culberson, Victoria 373A2-2-1-2	2
Forkedeer x Hairy Culberson, Victoria 374A1-3-1-1	2-
Fulwin C.I. 3168	2
Lee C.I. 2042	2
Lee, Victoria x Forkedeer[2] C.I. 5848	2+
Letoria C.I. 3392	3
Mustang C.I. 4660	1+
Nemaha x Forkedeer 439A1-47	3
Pioneer C.I. 3427	2-
Red Rustproof C.I. 1815	1+
Traveler C.I. 4206	2-
Victoria, Richland x Forkedeer 3812A1-1-2-1	2
Winter Turf C.I. 3296	1
Wintok C.I. 3424	2+
Woodward Sel. C.I. 4829	3
Woodward Sel. C.I. 5106	2+

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2. Murphy, H. C. 1952. Reaction of oat varieties and selections to Septoria black stem and powdery mildew. National Oat Newsletter 2 (1951): 32-33.

PURDUE UNIVERSITY AGRICULTURAL EXPERIMENT STATION, LAFAYETTE, INDIANA, AND DIVISION OF CEREAL CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AGRICULTURAL RESEARCH ADMINISTRATION, UNITED STATES DEPARTMENT OF AGRICULTURE, COOPERATING.

SEPTORIA NODORUM ATTACKING WHEATS IN THE SOUTHEASTERN COASTAL PLAIN

R. W. Earhart and D. D. Morey

The wheat glume blotch fungus, Septoria nodorum (Berk.) Berk. (identified by G. F. Weber) has been found infecting wheat extensively in the Southeastern Coastal Plain. This is the first observation of this fungus in Florida and the disease is being watched with special interest. Infections, which were first noted about February 15, appear to be heaviest on certain selections from the crosses Gullen^2 x Gaza and Bobin^2 x Gaza grown in preliminary tests. Wheats of the Atlas 50, Atlas 66, and Coastal types possess considerable resistance, although light infections have been found in these varieties at all locations.

Infections were found on the basal portion of the leaf blades, on the sheaths, and in some cases as a brown coloration on the culms beneath the sheaths. The lesions associated with these infections are characteristically darker and more distinct than previously reported for this disease. In early stages of development the lesions are slate-gray to greasy black in color, similar to those caused by Helminthosporium sativum on wheat and barley, or Septoria avenae on oats, Eventually these lesions become light-brown or tan. Pycnidia are initiated in infected tissue in the stomatal chambers and they develop and mature in these lesions after the death of the host tissues. Lesions range in size from pin-point spots to oval or lens-shaped lesions 6 to 7 centimeters long, frequently being as wide as the leaf blade. Glume infections have been noted on some of the wheats that are now coming into full head.

U. S. DEPARTMENT OF AGRICULTURE, DIVISION OF CEREAL CROPS AND DISEASES, AND THE FLORIDA AGRICULTURAL EXPERIMENT STATIONS, GAINESVILLE

EXPERIMENTAL FORECAST OF WHEAT LEAF RUST IN OKLAHOMA FOR 1953

H. C. Young, Jr., D. F. Wadsworth, and J. R. Epperly

After the leaf rust forecast for the 1952 crop season was made (Plant Disease Reporter 36: 213, May 15, 1952) leaf rust (Puccinia rubigo-vera var. tritici) development proceeded according to prediction until about May 1, 1952. Then, for the first time since these forecasts have been made, rust development was almost completely retarded by a period of hot, dry winds. Subsequent development caused an estimated loss of about 5 percent of the crop, instead of the 7 to 10 percent originally predicted.

The summer and fall of 1952 in Oklahoma were the driest on record. The drought period extended well into November, preventing the germination of volunteer grain and delaying the seeding and emergence of the 1953 crop. Consequently, very little, if any, rust oversummered in the State, and no fall or winter infections were observed. Slide exposures indicated that spore showers during the fall and winter were extremely light.

Examinations of a rust susceptible wheat variety were started as usual at Stillwater February 1, and continued at 10-day intervals until April 1, 1953. No rust pustules could be found until April 1, when 122 pustules were counted on 1000 tillers. This may be compared with 4947 pustules on 1000 tillers at the same date in 1952.

During the period of March 28-31, an 1850 mile survey was made through the State. Not a single rust pustule was found in any of the fields or nurseries inspected.

The loss from leaf rust in the 1953 crop is expected to be less than one percent, compared to an average annual loss of approximately 5 percent. While wheat is estimated to be from one to three weeks late, the absence of rust in the southern sections of the State indicates that the crop will escape any appreciable damage. Some rust development will undoubtedly occur, particularly in very late fields in low areas where slight damage may be expected.

OKLAHOMA AGRICULTURAL AND MECHANICAL COLLEGE, STILLWATER

STEWART'S DISEASE PROSPECTS FOR 1953

G. H. Boewe

In 1953, Stewart's disease, or bacterial wilt (Bacterium stewartii), of corn probably will occur over most the State and be more destructive much farther north in Illinois than it was in the summer of 1952. It probably will be more destructive than it was in the summer of 1950.

FIGURE 1

Since this is the warmest winter in Illinois since that of 1931-32, damage and loss from Stewart's disease may possibly be the heaviest it has been in the past 21 years.

Two principal types of damage to corn are produced by Stewart's disease: an early-season wilt and death of plants, especially in sweet corn, and late-season leaf blight, which may be severe enough to cause death of plants, on both susceptible sweet corn and field corn. The wilt and leaf blight phases of the disease are expected to appear on susceptible varieties of corn in Illinois as shown by the table below.

	Region	Early-season wilt	Late-season leaf blight
I.	Extreme north west	None	None or a trace
II.	Extreme north	Absent or nearly so	Light to moderate
III.	North	Absent or nearly so	Moderate to severe
IV.	North central	Light to severe	Severe
V.	South half	Destructive	Severe

The five regions listed in the Table are delineated on the map (Fig. 1). These regions have been determined on the basis of winter temperature indexes (the sum of the mean temperatures of December, January, and February of the past winter) calculated from the records of 95 weather reporting stations distributed throughout the State. The data were obtained from the U. S. Weather Bureau office at Springfield. The range of indexes and an average index for each region are shown in the following table:

Region	Range of indexes	Average index
I	77.7	
II	81.6 - 87.0	83.8
III	85.4 - 89.8	87.2
IV	87.7 - 98.9	94.4
V	97.7 -125.7	110.4

As indicating the future occurrence of Stewart's disease, these indexes appear to have significance, as follows:

If the index is	Early season wilt probably will be	Late season leaf blight probably will be
100 or more	Destructive	Severe
Between 90 and 100	Light to severe	Severe
Between 85 and 90	Absent or nearly so	Moderate to severe
Between 80 and 85	Absent or nearly so	Light to moderate
Below 80	Absent or nearly so	A trace, at the most

This forecast on Stewart's disease, the fifth to be made for Illinois, is based on the close relationship that appears to exist between the amount of disease which develops during the summer and the temperature of the preceding winter. The development of both phases of the disease on sweet corn during the past four years coincided very closely with the predictions made in the first four forecasts. The development of the leaf blight phase on field corn has also coincided very closely with our previous predictions.

SECTION OF APPLIED BOTANY AND PLANT PATHOLOGY, ILLINOIS STATE NATURAL HISTORY SURVEY, URBANA

EFFECT OF INCREASED PHOTOPERIOD ON EGG MASS PRODUCTION BY THE ROOT-KNOT NEMATODE, MELOIDOGYNE INCOGNITA (KOFOID AND WHITE) CHITWOOD[1]

A. C. Tarjan and B. E. Hopper[2]

With several investigators in quest for cheaper, more effective, and more versatile nematicides than those already developed, the use of the root-knot nematodes as test organisms is an established routine. Simplicity of cultural procedures and the relatively short time required for completion of the life cycle enhance the value of these nematodes for this purpose. Various soil factors have been pointed out as affecting the incidence of root knot (2), among which the effect of temperature has been shown to be of paramount importance (1, 4). Certain investigators (3, 4), in working with root knot of tomato, increased the photoperiod of infected test plants by use of artificial light, but did not explain the significance of this procedure. Although increasing the photoperiod may result in larger and more vigorous plants, the effect of extra light on the reproductive activities of the nematode has not been determined. This investigation evaluates the effect of increased photoperiod on egg mass production by root-knot nematodes at two different greenhouse temperatures.

Methods

Twenty-four five-week old tomato plants, Lycopersicon esculentum hort. var. Valiant, each growing in a 5-inch pot in a potting mixture of 1 part loam to 1 part sand, were inoculated with five egg masses containing infectious, second-stage larvae of the root-knot nematode, Meloidogyne incognita. Two groups of six plants each were placed in a section of the greenhouse exposed to an average temperature of 66° F (58.8-73.2°), while the remaining two groups of six plants each were placed in a section exposed to an average temperature of 81.8° (73.3-90.3°). One group in each section received the normal photoperiod of 9 to 11 hours of daylight, while the other group received the normal photoperiod plus sufficient illumination from 150 Watt incandescent lamps delivering 800 foot candles at the apex of the plants to amount to at least 16 hours of illumination each 24-hour period. This experiment was started on November 14, 1952 and terminated on January 5, 1953.

In harvesting, the root system of each plant was first washed gently in water to eliminate soil and debris, after which the fresh weight of roots was recorded. The root system of each plant was then finely chopped and thoroughly mixed after which a representative sample of 500 mg. was placed in a shallow layer of water in a Syracuse watch glass and examined for the presence of adult female nematodes and egg masses. Having obtained counts from each sample, values were then computed on the basis of root weight of each plant in order to estimate the total number of females and egg masses per plant. Values for the nematode reproductive index for each plant were obtained by dividing the total number of egg masses per plant by the total number of females.

Results

Differences in reproductive activity between nematodes infecting plants grown at 81.8° and 66.0° F were significant at odds of 99 to 1 (Table 1). Although length of photoperiod had no significant effect on total numbers of females or egg masses formed in plants exposed to an average temperature of 66.0°, such differences between plants exposed to an average temperature of 81.8° were significant at odds of 99 to 1. Length of photoperiod had no effect on nematode reproductive indexes of plants grown at the same average temperature. Reproductive indexes, however, did differ significantly between plants grown at the different temperatures, presumably because many of the mature females counted on roots of plants grown at 66.0° had not yet formed egg masses at this lower temperature.

From these data it can be concluded that an abundant supply of root-knot egg masses can be obtained in the shortest possible time by growing inoculated plants in warm temperatures under an increased photoperiod.

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[1]Contribution No. 824 from the Rhode Island Agricultural Experiment Station, Kingston.

[2]Assistant Research Professor and Student Assistant respectively, Department of Plant Pathology - Entomology.

Table 1. The effect of increased photoperiod on oviposition by Meloidogyne incognita at two greenhouse temperatures.

Average temperature	Photoperiod	Total females per plant	Total egg masses per plant	Reproductive index
81.8°F	Normal	1,325[a]	1,209	0.91
	Increased	2,083	1,873	0.90
66.0°F	Normal	64	43	0.64
	Increased	155	90	0.58
	LSD .05	388	347	0.18
	LSD .01	537	480	0.25

[a] Each value represents the average count from 6 replicate plants.

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RHODE ISLAND AGRICULTURAL EXPERIMENT STATION, KINGSTON

GEOGRAPHICAL DISTRIBUTION OF SOME MELOIDOGYNE SPP. IN ISRAEL[1]

A. C. Tarjan

Although numerous reports of root-knot nematode infestation in Israel have been published in recent years (1, 2, 3, 4, 5), these accounts identify the parasites collectively as Heterodera marioni, a designation which has been superseded by the generic name Meloidogyne consisting of several species (6). Through the cooperation of Dr. G. Minz, Division of Plant Pathology, Agricultural Research Station, Rehovot, Israel, a number of preserved nematized root samples from various parts of Israel were submitted for identification.

A number of adult female nematodes were dissected from roots of each sample and the section of the body bearing the anal plate (perineal) pattern was cut and mounted in clear lactophenol. Designation of the species in each case was based on characteristics exhibited by the anal plate patterns as well as measurement of certain structures in the female head. A minimum of five nematodes from each sample was examined before a population was identified (Table 1). Dr. J. N. Sasser kindly verified several of the identifications made.

Table 1. Meloidogyne species found parasitizing various hosts in Israel.

Sample No.	Host Plant	Locality	Nematode Species
1	Lycopersicon esculentum Mill.	Rehovot	M. javanica (Treub) Chitwood
4	Lycopersicon esculentum	?	M. incognita (Kofoid & White) Chitwood
6	Nicotiana tabacum L.	Qubeiba	M. javanica
7	Beta vulgaris var. crassa L.	Ein Tzurim	M. javanica
8	Lycopersicon esculentum	B'er Yaqov	M. javanica
9	Antirrhinum majus L.	Rehovot	M. javanica
11	Angelica officinalis Hoffm.	Rehovot	M. javanica
12	Valeriana officinalis L.	Rehovot	M. incognita
13	Lycopersicon esculentum	Ramleh	M. javanica
14	Armoracia lapathifolia Gilib	Gibton	M. javanica
15	Petroselinum sativum Hoffm.	Rehovot	M. incognita
16	Portulaca oleracea L.	B'er Yaqov	M. javanica
17	Cucumis sativus L.	B'er Yaqov	M. javanica
18	Chenopodium sp.	B'er Yaqov	M. javanica
20	Solanum nigrum L.	Rehovot	M. arenaria (Neal) Chitwood and M. javanica
21	Amaranthus caudatus L.	Rehovot	M. javanica
23	Solanum melongena L.	Rehovot	M. javanica
24	Solanum tuberosum L.	Rehovot	M. javanica
25	Olea europaea L.	Acre	M. javanica
26	Ricinus communis L.	Rehovot	M. javanica
27	Impatiens balsamina L.	Rehovot	M. javanica
28	Lycopersicon esculentum	?	M. arenaria
29	Lycopersicon esculentum	?	M. javanica
30	Beta vulgaris var. crassa	Salameh	M. sp.
31	Lycopersicon esculentum	Kfar Blum	M. javanica
32	Prunus amygdalus var. amara Batsch.	Wilhelma	M. javanica
33	Valeriana italica L.	Rehovot	M. hapla Chitwood
34	Solanum tuberosum	B'er Yaqov	M. javanica

[1]Contribution No. 823 from the Rhode Island Agricultural Experiment Station, Kingston.

Of interest in Table 1 is the widespread distribution of Meloidogyne javanica as well as the sporadic occurrence of other Meloidogyne species. Anal plate patterns of root-knot nematode females in sample No. 30 were decidedly different than any of the patterns exhibited by known species of Meloidogyne and will be studied more thoroughly.

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RHODE ISLAND AGRICULTURAL EXPERIMENT STATION, KINGSTON

PROGRESS REPORT OF NATIONAL SCREENING COMMITTEE FOR DISEASE RESISTANCE IN THE TOMATO FOR 1952[1]

Summarized by Leonard J. Alexander, Ohio Agricultural Experiment Station, Wooster, Ohio, and M. M. Hoover, Primary Plant Introduction Station, Ames, Iowa

Preliminary Report on Screening Wild Species for Disease Resistance

This preliminary report on the work accomplished in 1952 is by necessity brief. No attempt is made to publish the results in detail. Rather, this is a summary, but in certain instances the accessions showing the highest type of resistance found to diseases are listed.

The more complete report with all the data should follow after the results of 1953 have been secured. It is planned to publish in the complete report all the data for the 144 accessions of the wild species. The complete data will enable interested persons to select breeding stock best suited to their needs.

It has been observed by some cooperators that many of the seed stocks sent to them for the screening trials were not homozygous. This lack of homozygosity was noted when the seed was multiplied, but it was not deemed wise to attempt to pureline the accessions. It was felt rather, that the best procedure was to attempt to maintain the lines as they were in order to preserve whatever germ plasm they possessed. Then, if a line was found to possess resistance to a certain disease the individual worker could pureline the accession for the character with which he wished to work. One exception to this procedure was followed, i.e., in those cases where obvious mixtures were present no seed was saved from the contaminants.

Tomato accession numbers referred to in this report (Table 1) are those assigned to the tomato stocks by the Division of Plant Exploration and Introduction, Bureau of Plant Industry, Soils, and Agricultural Engineering, Beltsville, Maryland.

The separate reports for each disease are listed in alphabetical order.

Alternaria solani: Three reports were submitted on the screening for Alternaria resistance, by W. S. Porte, C. F. Andrus, and D. F. Jones. Porte and Andrus both secured good degrees of infection. The incidence of infection in Jones' tests was very light and his results are not included in this summary. Porte worked with the early blight phase of Alternaria; Andrus with the collar rot phase. Readings for resistance to the two diseases are in very close agreement and are fairly complete for the 144 accessions of wild species distributed for this screening program.

A high order of resistance is reported, in classes "1" and "2", for accessions in each of the five species, except Lycopersicon glandulosum for which the highest rating was "3". L. hirsutum, accession 126445, and L. esculentum x L. pimpinellifolium, accession 112835, show greatest resistance with ratings of "1". Twenty-seven accessions in the four species have a class rating of "2". These 27 accessions by species are: L. hirsutum -- 127826 and 127827; L. hirsutum variety glabratum -- 129157 and 134417; L. peruvianum -- 126926, 129146; L. pimpinellifolium -- 112215, 126932, 126940, 126947, 126949, 127806, 127833, and 128639; species crosses, L. esculentum x L. pimpinellifolium -- 118403, 118405, 118407, 118784, 124132, 126948, 129030, 129116, 129143, 155375, 185688, 185689, and 187002.

Anthracnose (Colletotrichum phomoides): Of the three members of the committee for screening for anthracnose resistance, only A. D. Hoadley, was able to obtain satisfactory levels of infection. Certain of the lines reported by him did not produce sufficient fruit to give satisfactory tests. Of the 144 lines distributed, he reported on 74. His anthracnose ratings were secured from artificially inoculated field-grown plants.

No results were secured in the species Lycopersicon hirsutum, L. hirsutum var. glabratum, or L. peruvianum. In L. pimpinellifolium, none of the accessions received a rating of "1" Two accessions, 126927 and 126953, rated "2". In L. glandulosum, a rating was obtained on only the one accession, 126441, which rated a "2". In the species crosses, one accession, 114038, received a rating of "1", and three accessions, 129027, 155379, and 166365, received ratings of "2". All other accessions received ratings of "3" or more.

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[1]Ohio Agricultural Experiment Station, Journal Article No. 27-53.

Cooperators on National Screening Trials for Disease Resistance in the Tomato

ALTERNARIA
C. F. Andrus*
C. E. Cox
A. L. Harrison
D. F. Jones
M. L. Odland
W. S. Porte
S. A. Wingard

ANTHRACNOSE
A. D. Hoadley
C. A. John
M. B. Linn*

BACTERIAL WILT
W. S. Barham*
D. E. Ellis
Donald C. McGuire

CLADOSPORIUM LEAF MOLD
E. F. Guba
E. A. Kerr*

CRACK RESISTANCE
G. B. Reynard*

FROST TOLERANCE
I. L. Nonnecke*

FUSARIUM
L. J. Alexander
James M. Epps
Wm. M. Epps*
A. L. Harrison
J. W. Lesley
J. T. Middleton

Fusarium, cont.
W. S. Porte
F. C. Stark, Jr.
Miriam C. Strong
V. M. Watts

NEMATODES
C. E. Cox
A. L. Harrison
Donald C. McGuire
F. A. Romshe
A. L. Taylor*

PHOMA
J. M. Walter*

PHYTOPHTHORA (Late blight)
W. S. Barham
Reiner Bonde
B. A. Conover
D. E. Ellis
E. L. Felix
M. E. Gallegly, Jr.*
K. M. Graham
M. L. Odland
W. T. Schroeder
J. M. Walter
S. A. Wingard

SCLEROTIUM ROLFSII
J. E. Denman
E. L. Moore*

SEED INCREASE
G. W. Bohn
W. P. Cowley

Seed increase, cont.
Donald C. McGuire
W. J. Virgin

SEPTORIA
A. A. Cook
W. J. Hooker*
R. W. Samson
Mark L. Tomes
E. P. Lana

SPOTTED WILT
Donald C. McGuire*

STEMPHYLIUM
J. F. Darby
A. D. Hoadley
Donald C. McGuire
R. W. Samson*
Mark L. Tomes

TOBACCO ETCH
J. M. Walter*

TOBACCO MOSAIC
L. J. Alexander
S. P. Doolittle*
Donald C. McGuire
R. K. Soost
H. H. Thornberry
S. G. Younkin

VERTICILLIUM WILT
O. S. Cannon*
L. G. Denby
J. W. Lesley
J. T. Middleton
W. J. Virgin

Bacterial Wilt (Xanthomonas solanacearum): At the time of the writing of this report, only the work of D. E. Ellis and W. S. Barham are available. Donald C. McGuire has tests underway but the results are not yet complete.

Of the 144 accessions Ellis and Barham report the following as being worth testing further: In the species Lycopersicon peruvianum -- 126946; in the species L. pimpinellifolium -- 112215, 126924, and 143522; in the species L. glandulosum -- 126444; and in the species crosses, L. pimpinellifolium x L. esculentum -- 108245, 126912, 129027, and 147609. Concerning these accessions, Ellis states:

> "These lines all appeared to show possible low levels of resistance to Pseudomonas solanacearum in one greenhouse inoculation test."

It is interpreted from this that none of the accessions showed a high level of resistance, but that these nine possess a certain degree of resistance.

Cladosporium Leaf Mold (C. fulvum): E. A. Kerr of the Vineland Station of Ontario was the only member of the committee to screen the accessions for Cladosporium leaf mold resistance. He submitted a rather complete report for resistance to race 6. The data were obtained on

Table 1. Tomato accessions for National Screening Program, 1952.

Lycopersicon species Accession no.	Lycopersicon species Accession no.	Lycopersicon species Accession no.
1. hirsutum	3. (cont.)	5. (cont.)
126445	126432	118784
127826	126433	118786
127827 (10 seeds)	126436	119104
199381 (10 seeds)	126924	119214
	126925 (20 seeds)	124132
hirsutum var.	126927	126437
glabratum	126931	126905
126449	126932	126912
129157	126933	126915
134417	126934	126923
134418	126937	126943
	126938	126948
2. peruvianum	126939	127799
126431	126940	128194
126926	126941	128664
126929	126947	129021
126930	126949	129027
126935	126952	129028
126944	126953	129030
126945	126954	129062
126946	127805	129074
127830	127806	129089
127831	127807	129090
127832	127833	129105
128643	128639	129108
128645	129156	129116 (24 seeds)
128646	133542	129143
128647	143522	129148
128648	143679	129154
128649	144955	129155
128650	190256	129686
128651		135022
128652	4. glandulosum	135842
128653	126434	143680
128655	126440	146089
128656	126441	147609
128657	126443	147635
128659	126444	152043
128660	126448	155368
128661 (25 seeds)	199380 (10 seeds)	155369
128663		155371
129135	5. Species cross	155375
129146	esculentum x	155378
129149	pimpinellifolium	155379
129152	108245	158164
	112835	158166
3. pimpinellifolium	114038	158167
79532	114967	159007
110595	118403	163246 (26 seeds)
112215	118405	166365
124039	118407	185688
126430	118409 (20 seeds)	185689
		187002

seedlings grown in flats in the greenhouse. In the species Lycopersicon hirsutum, accession 127826 rated "1", accessions 126445 and 127827 rated "2". In L. hirsutum var. glabratum accession 134417 rated "2", and some of the others "3" or more. In the species L. peruvianum a great deal of resistance was found. Only accession 128659 received a rating of "3". Of the others, 24 rated "1", and 7 rated "2". In L. pimpinellifolium, the accessions 112215, 126934, 126938, 126940, and 143679 rated "1"; 126433, 126925, 126937, 126947, and 129156 rated "2"; 126931 and 126954 appeared to segregate for resistance and susceptibility; and other accessions rated "3" or more. In L. glandulosum, accessions 126434, 126440, 126448, and 199380 rated "1". Two accessions, 126441 and 126443, appeared to segregate for resistance and susceptibility. The other two accessions received ratings of "3" or more. In the species crosses, accessions 114967 and 129148 rated "1"; 129021 and 147635 rated "2"; 15 additional accessions segregated for resistance and susceptibility; and the remainder rated "3" or more.

Kerr made the following observation:

> "From these screening trials there appear to be several genes for resistance to C. fulvum which are distinct from those which have been identified. Plant breeders have a large number of stocks from which a breeding program for resistance to C. fulvum can be initiated."

Crack Resistance: G. B. Reynard of the Campbell Soup Company served alone on a committee to classify accessions for crack resistance. Results were recorded by Paul V. V. Weber. The notes were taken on two plants. A number of these accessions bore no fruits and could not be reported on. This was the case with Lycopersicon hirsutum which did not fruit under the conditions at Riverton, New Jersey. In the species L. hirsutum var. glabratum, L. peruvianum, and L. glandulosum, no cracking was observed. In L. pimpinellifolium very little cracking was noted, but accession 126924 showed a slight amount of concentric cracking, while accession 126941 and 144955 showed severe concentric cracking. In the species crosses, some cracking did occur. However, quite a few of the accessions were free of cracking. These accessions are: 108245, 112835, 114038, 114967, 118403, 118407, 118784, 118786, 126948, 127799, 128194, 129027, 129028, 129062, 129090, 129105, 129143, 129155, 143680, 147609, 152043, 155375, 158164, 158166, 159007, 163246, and 166365.

Frost Tolerance: I. L. Nonnecke, Agricultural Experiment Station, Lethbridge, Alberta, Canada, was the only worker testing for frost tolerance. Unfortunately, no spring frost occurred after plants were set in the field. Fall weather also was mild and when frost came it was severe and did not allow differentiation between accessions. It is planned to repeat these tests in 1953.

Fusarium Wilt (F. oxysporum f. lycopersici): Of the workers who are cooperating for screening for Fusarium wilt, results were secured from Porte, Epps, Harrison, Strong, Watts, Lesley, and Alexander. The first six screened for resistance to the race common in their area, whereas Alexander screened for resistance to two races. Best resistance to Fusarium wilt was reported with the species Lycopersicon peruvianum, in which resistance to all races is found. It is very difficult, however, to cross this species with the domestic species. In general, to be successful crossing has to be done by embryo culture. A high type of resistance was found in L. pimpinellifolium also. All investigators reported accession 79532 to be highly resistant to the common race, although Alexander showed it to be susceptible to race 2. Other accessions showing high resistance to the common race are 124039, 126937, 128639, and 129156. Only accessions 124039, 126931, 126954, and 143679 showed a type "2" resistance to race 2. A high type of resistance to both races existed in all accessions of L. glandulosum. In the species crosses, accession 129155 showed the highest type of resistance to the common race and to race 2.

Epps very ably summarized the conclusions that may be drawn from screening for Fusarium resistance, as follows:

> "1. These data indicate that few if any of the accessions are completely immune to the races of the pathogen tested. Breeders should test each plant individually before using it as a source of resistance to Fusarium wilt.
>
> "2. Several accessions appeared to be susceptible to Alexander's race 2 (Culture A-1-3) and resistant to race 1 (represented by Alexander's culture T-6 and the cultures of the other cooperators). These include 79532, 126937, 126952, 126953, 127833, 128639,

129156, 129027, 143680, and the variety Pan America which was developed from crosses involving 79532.

"3. It appeared that there are other races among the pathogen cultures used, at least one, in addition to the two used by Alexander. Culture A-1-3 may be separated from the other cultures used by its reaction on the lines listed above. The other cultures may be separated into two groups by their reaction on L. hirsutum var. glabratum. The four accessions in this botanical variety were rated as resistant (Rating 1 or 2) by Harrison, Strong, and Epps and as susceptible (3, 4, or 5) by Porte, Watts, and Lesley. The differences were too clear cut to be ignored. Further study will be necessary to solve this problem.

"4. All accessions in the three species L. hirsutum, L. peruvianum, and L. glandulosum contained some wilt immune individuals, but no accessions proved completely immune in all tests.

"5. The species L. pimpinellifolium and the crosses of L. esculentum and L. pimpinellifolium contained some lines resistant to wilt, but most lines were wilt susceptible.

"6. Most of the 144 accessions need not be retested. Those checked in the committee report and possibly a few others should be rechecked either because the cooperators failed to agree or the number of plants tested was too small to give satisfactory evaluation."

Nematodes: Of the committee screening for nematode resistance, data were secured from F. A. Romshe, A. L. Harrison, and A. L. Taylor. C. E. Cox considered his field tests at College Park, Maryland, unsatisfactory and did not report. D. C. McGuire's tests at Honolulu were not finished at the time of writing this report, but will be available later.

The root knot nematode was formerly considered as a single species. However, it has since been divided into several groups. Taylor states in his report:

"Until 1949, all of the root knot nematodes were grouped under the name Heterodera marioni, and it was generally believed that the host habits were more or less uniform for all locations, even though there was considerable evidence to the contrary. Christie (1944) demonstrated by controlled experiments that root knot nematodes from different locations did indeed differ in their reactions to various host plants. Chitwood (1949) was able to distinguish five species and one variety of root knot nematodes by morphological characters and transferred all of these to the genus Meloidogyne. Later he described an additional subspecies. Since that time some progress has been made in identification of the species and data on their distribution has gradually accumulated, but it will be many years before the work is finished. At this time, it appears that at least six of the seven described forms occur in cultivated soil in the United States as follows:

"Meloidogyne incognita is fairly common in most of the warmer regions of the country.

"M. incognita var. acrita appears to be most common form in the southern and western States.

"M. arenaria has been collected from scattered locations in the South.

"M. arenaria subsp. thamesi is apparently fairly common in Florida and has been found in other parts of the country.

"M. javanica has been collected from scattered locations in the United States.

"M. hapla is the most common root knot nematode in the northern States, but occurs in scattered locations in many parts of the United States.

"The remaining described species, M. exigua, has been collected in the United States only at the New York Botanical Garden, and also from coffee in Central and South America."

Taylor's data involved the species Meloidogyne incognita, M. incognita var. acrita, M. arenaria, and M. javanica; at Stillwater, Romshe worked with M. incognita var. acrita. The species with which Harrison worked were not listed.

The highest order of resistance to nematodes was found in the species Lycopersicon hirsutum, L. hirsutum var. glabratum, L. peruvianum, and L. glandulosum. Very little resistance was found in L. pimpinellifolium or the species crosses. Of the species which showed some resistance, the greatest was found in L. peruvianum. Two accessions of this species, 128657 and

128653, showed the highest degree of resistance to the groups of the root knot nematode worked with. Taylor did not consider that accessions with ratings of 3, 4, or 5 were worth further testing. He states:

> "The above data are considered as being preliminary only since there is always a possibility that low readings on susceptibility may be due to other causes than resistance in small scale tests. Ratings of 3, 4, or 5, on the other hand, can be taken as indicating that the accession is probably not desirable breeding material. This would appear to eliminate Lycopersicon pimpinellifolium and crosses of this species with L. esculentum from consideration. L. hirsutum var. glabratum showed resistance only to M. arenaria at Beltsville. L. glandulosum and L. peruvianum accessions were variable, both as to reactions of the individual accessions to the various species of nematodes and as to reactions of the different accessions to the same nematode species."

In 1953 additional tests are planned for all accessions which showed ratings of 1 or 2 in any test.

Phoma destructiva: J. M. Walter served as a one-man committee to classify the accessions for Phoma resistance. He used six classes to record the degree of resistance, "0" being immune and "5" most susceptible. In addition to the accessions submitted, he included accession 115201, a Lycopersicon esculentum-type species. No resistance was observed in L. hirsutum, L. hirsutum var. glabratum, L. peruvianum, or L. pimpinellifolium. Two accessions in L. glandulosum, 126448 and 199380, showed some resistance, rated as a "2". Four accessions in the species crosses showed a "2" type of resistance. These are 119214, 126905, 128664, and 129028. The one accession of L. esculentum-type species, 115201, gave a "1" type of reaction. Walter made selections from five plants in accession 115201, which he considers to possess a very good type of resistance.

Phytophthora infestans: Of the committee to screen for late blight resistance, M. E. Gallegly, Jr., K. M. Graham, and Reiner Bonde were able to submit reports. Bonde tested only the crosses, whereas Graham and Gallegly tested all the 144 lines submitted. They did not find sufficient resistance in the species Lycopersicon hirsutum, L. hirsutum var. glabratum, L. peruvianum, or L. glandulosum to work with. In L. pimpinellifolium, accessions 110595 and 190256 showed some resistance. In the species crosses they found accessions 108245, 114038, 114967, 119214, 124132, 126437, 129089, 129108, 163246, and 166365 to be resistant. In all cases, they worked with the common race of Phytophthora infestans. Gallegly made plant selections from certain of these accessions, and in addition has plant selections from some other material. As soon as possible this seed will be multiplied, given P.I. numbers, and sent out to those interested in Phytophthora resistance.

Sclerotium rolfsii: No report was submitted for 1952.

Seed Multiplication: W. J. Virgin of the California Packing Corporation secured seed of a number of the lines of which we are in short supply. This was very much appreciated. If anyone else can get sufficient seed of some of the lines that fruit with difficulty for us, it would indeed be helpful. Our stocks are lowest in the species L. hirsutum.

Septoria lycopersici: Of the committee working on Septoria resistance, data were secured from M. L. Tomes and R. W. Samson of Indiana, A. A. Cook of South Dakota, and W. J. Hooker and E. P. Lana of Iowa. The latter two included in their trials a large number of Lycopersicon esculentum accessions that are not mentioned in this report at this time. Cook's readings for resistance were highest. It is possible that conditions for infection in South Dakota were not so favorable as in Iowa and Indiana, and of course another possibility is that Cook worked with a weaker pathogen. None of the lines showed a resistance as high as "1". Resistance of rating "2" was found in the species L. hirsutum, L. hirsutum var. glabratum, L. peruvianum, and L. glandulosum. One accession of the species L. pimpinellifolium, 143679, was given a rating of "2". Two accessions of the species crosses, 155378 and 155379, received ratings of "2" at Iowa, but "4" and "5" at Indiana and South Dakota. It is rather noticeable that there is considerable indication of variation or segregation within the accessions showing some resistance. Perhaps this could be expected as there is some evidence that some of these species are largely self-sterile but cross-fertile. This condition has been reported for the species L. peruvianum.

Higher orders of resistance are needed and it is possible that some improvement could be obtained by selfing some of the more resistant lines.

Spotted Wilt Virus (Lethum australiense, Lycopersicon virus 3): D. C. McGuire who served alone on the committee to screen for spotted wilt resistance, encountered difficulties in the 1952 program and was unable to make a report. He plans on completing this report for 1953.

Stemphylium solani: Of the committee screening for Stemphylium resistance, only the work of the Campbell Soup Company was satisfactory, other members failing to secure infection in 1952. From the readings taken by A. D. Hoadley, accession 127826 of the species Lycopersicon hirsutum showed a "1" for resistance. In the species L. hirsutum var. glabratum, accession 126449 also showed a "1" for resistance. In L. peruvianum, accession 129152 received a "1" rating; 128645, 128651, and 129149 rated "2"; and the other accessions in this species rated "3" or more. None of the accessions in L. glandulosum showed ratings of "1" or "2". In L. pimpinellifolium, three accessions, 126432, 126934, and 126949 rated "1". However, accession 126934 segregated for resistance and susceptibility and some plants were classified in the "5" rating. Accessions 126939 and 190256 received ratings of "2". The other accessions in this species were classed as susceptible. In the species crosses, two accessions, 114967 and 185689, showed ratings of "1"; six accessions, 112835, 129074, 129116, 166365, 185688, and 187002, rated "2"; and other accessions were rated "3" or more.

Tobacco Etch Virus (Marmor erodens, Nicotiana virus 7): J. M. Walter served as a committee to test for tobacco etch resistance. He tested the P.I. accessions that he had on hand; with a few exceptions, these did not include the species crosses. He reports that there was possibly some indication of resistance in accession 183692, Lycopersicon esculentum.

Tobacco Mosaic Virus (Marmor tabaci, Nicotiana virus 1): S. P. Doolittle's report on screening for mosaic is as follows:

"In five trials in the greenhouse species of Lycopersicon were inoculated with Johnson's type strain of green tobacco mosaic virus (from H. H. McKinney). In another trial a small number of plants were grown in the field where they were subject to natural infection by unidentified virus strains. In the Ohio trials a second series of plants were inoculated with McKinney's strain (BSY) of yellow tobacco mosaic virus. In trials at Beltsville plants showing no symptoms after inoculation with the green strain were re-inoculated with the yellow strain of the virus.

"The outstanding result of the trials was the evidence of segregation for mosaic resistance in 27 accessions of Lycopersicon peruvianum. Some plants showed no virus symptoms and no evidence of masking of the virus was found when they were tested by inoculations on Nicotiana glutinosa. This was true of plants inoculated with either the green or the yellow strain of virus. The P.I. numbers of these accessions are 126431, 126926, 126929, 126930, 126935, 126944, 126945, 126946, 127830, 127831, 127832, 128643, 128645, 128646, 128647, 128648, 128650, 128651, 128652, 128653, 128655, 128656, 128657, 128659, 128663, 129135, and 129149.

"Some cooperators reported segregation in 4 P.I. accessions of L. hirsutum (126445, 127826, 127827, and 199381), but the number of resistant individuals appeared to be much less than in the accessions of L. peruvianum. Resistant plants were found also in accessions classed as L. glandulosum, but it is hard to evaluate the findings because there were a number of plants typical of L. peruvianum in these accessions.

"No resistance was noted in trials of 35 accessions of L. pimpinellifolium. One other accession in this group (P.I. 143679) was typical of L. peruvianum and segregated for resistance. All 4 accessions of L. hirsutum var. glabratum and all 61 accessions of crosses of L. esculentum x L. pimpinellifolium proved susceptible.

"The Ohio trials indicated that more plants in segregating accessions were susceptible to the green strain of tobacco mosaic virus than to the yellow.

"Cooperators reporting the results of screening trials to tobacco mosaic virus were as follows: L. J. Alexander, Department of Botany and Plant Pathology, Ohio Agricultural Experiment Station, Wooster, Ohio; S. P. Doolittle and F. S. Beecher, Division of Vegetable Crops and Diseases, Plant Industry

Station, Beltsville, Maryland; R. K. Soost, Division of Plant Breeding, Citrus Experiment Station, University of California, Riverside, California; H. H. Thornberry, M. B. Linn, and W. A. Huelsen, Department of Horticulture, Illinois Agricultural Experiment Station, Urbana, Illinois; and Paul V. V. Weber, Department of Agricultural Research, Campbell Soup Company, Riverton, New Jersey."

Verticillium Wilt: No reports were received on Verticillium wilt.

Conclusions

From the results secured in 1952 and those anticipated for 1953, it is expected that valuable germ plasm for disease resistance in the tomato either has already been or will be found. In fact, the 1952 results demonstrated that we possess a great deal of valuable germ plasm, especially for resistance to diseases, that we did not know about before.

In order to exploit this work to the fullest extent, it will be necessary to hybridize the accessions of Lycopersicon peruvianum with varieties of the domestic species. A large number of such crosses will be attempted in Ohio in 1953, using the embryo culture technique. It is true that resistance to some diseases has been found in accessions that hybridize readily with varieties of the domestic species, which of course simplifies the problem of transferring disease resistance to commercial varieties. It is important, however, that crosses be secured with accessions that hybridize with difficulty with varieties of the domestic species.

NATIONAL SCREENING COMMITTEE FOR DISEASE RESISTANCE IN THE TOMATO

UNUSUAL RECORDS AND OTHER BRIEF NOTES ON PLANT DISEASES

STEM-END STRIPES OF TOMATO FRUITS

By P. A. Young

FIGURE 1. Large red tomato fruit with yellow radial stem-end stripes. The cuticle was unbroken except for four short cracks.

Radiating stem-end stripes 1 to 4 centimeters long and 100 to 2000 microns wide were unusually abundant in tomatoes in June 1952 (Fig. 1). These stripes apparently were the beginning stage of fruit cracking through one or a few layers of epidermal cells. Below the unruptured cuticle, the injured stripe-cells were brick-shaped and 45 to 105 microns long. In contrast, the surrounding normal epidermal cells were polyhedral, 30 to 45 microns in diameter, and were not elongated. The stripes were dark green but became yellow as the fruits ripened. The latter color was due to the yellow cuticle. Some of the stem-end stripes ruptured the cuticle and appeared as corky-tan lines. Associated weather favored cracking of tomato fruits.

TOMATO DISEASE LABORATORY, TEXAS AGRICULTURAL EXPERIMENT STATION, JACKSONVILLE

VERTEBRAE-CRACK LINES OF TOMATO FRUITS

By P. A. Young

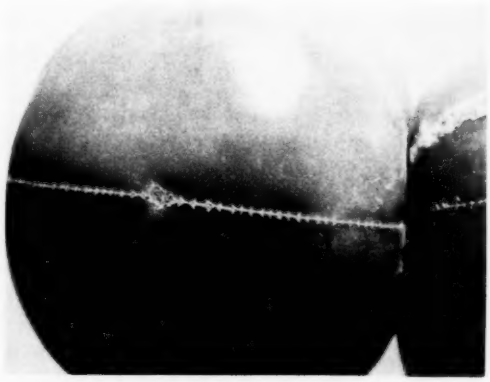

FIGURE 1. Vertebrae-crack line extending from the stem end to the blossom-end of a large tomato fruit. The crack line consisted of a chain of tan colored corky cracks.

A recently studied symptom of physiological cracking of tomato fruits was abundant in June 1952. It consisted of thin meridian lines that usually started near the stem end and extended toward or as far as the blossom end (Fig. 1). The meridian crack lines were 100 to 300 microns wide. Across them, the perpendicular cracks (usually with pointed ends) were 300 to 700 microns long and 350 to 750 microns apart. Thus, they resembled the vertebrae of a skeleton. These were jagged cracks in the cuticle through which air entered and discolored the epidermal cells. They became tan colored and corky in healing. Vertebrae-crack lines bordered some large deep blossom-end holes in tomato fruits. Evidently these lines started to develop in green fruits as small as 2 centimeters in diameter. They were noticed only in commercial varieties of green-wrap tomatoes with the U-allele for dark green tops.

Lenticel-like blemishes, described earlier, resembled vertebrae-crack lines in structure but differed in extending in various directions[1]. Pox symptoms resembled lenticels before and after rupturing the cuticle and apparently were symptoms of fruit cracking[2]. Hereditary stripes and pits in the peel resembled pox[3]. Weather in June 1952 favored the development of many different symptoms of fruit cracking.

TOMATO DISEASE LABORATORY, TEXAS AGRICULTURAL EXPERIMENT STATION, JACKSONVILLE

[1]Calvert, O. H. and P. A. Young. 1951. Lenticel-like blemishes on tomato fruits. Plant Dis. Reptr. 35: 120

[2]Ivanoff, S. S., and P. A. Young. 1940. Tomato fruit pox. Phytopath. 30: 343-345.
[3]Young, P. A. 1947. Horticultural characters of tomatoes. Texas Agr. Exp. Sta. Bul. 698.

MORE ABOUT THE GOLDEN NEMATODE IN PERU

By C. Bazán de Segura

During a survey to determine its presence and distribution in Peru, the golden nematode Heterodera rostochiensis Wr. was found on potato, Solanum tuberosum subsp. andigenum, in the Andes region. In Puno y Cuzco (South Peruvian Andes) E. García Pittman found the nematode associated with the following new hosts also[1]: olluco (Ullucus tuberosus), oca (Oxalis tuberosa), and wild quinua (Chenopodium apulifolium).

This year, in Huancayo (Central Peruvian Andes), Pittman discovered two additional new hosts, namely yellow potato (Solanum goniocalyx), and quinua cultivada (Chenopodium quinoa). Identification was confirmed from samples sent to this Station.

In 20 soil samples of 20 grams each, taken before cropping from a field in Huancayo not very heavily infested by H. rostochiensis, examination at this Station showed 685 old viable cysts, that is, 17 females for each 10 grams of soil.

CENTRO NACIONAL DE INVESTIGACION Y EXPERIMENTACION AGRICOLA DE LA MOLINA, LIMA, PERU

[1]Wille, J. E. and C. Bazán de Segura. 1952. La anguilula dorada, Heterodera rostochiensis, una plaga del cultivo de las papas, recien descubierta en el Peru. Peru, Centro Nac. Invest. y Exp. Agricola "La Molina", Lima. Bol. 48. 17 pp.

HEAD ROT OF BANANA RHIZOMES IN HONDURAS[1]

By H. H. V. Hord and R. S. Flippin

The appearance of an apparently new disease of banana rhizomes was first noted in a Standard Fruit and Steamship Company plantation in Honduras in June 1948. By March 1949 it was of common occurrence in the Company's plantations in the Aguan Valley. In new farms the disease first became prominent at about the time the first crop began to mature (12 to 15 months after planting), and continued through successive generations. "Head Rot" has been serious in new farms where arid soils have been put under cultivation for the first time and also in old banana farms flood fallowed for 18 months in an effort to eradicate the organism causing Panama disease.

Head rot can be most readily distinguished in these farms by the fallen pseudostems which are associated with the advanced stages of the disease. The pseudostem falls as the result of a break in the banana rhizome or "head" at a point near the ground level. The break is associated with an area of decay that weakens the structural strength of the rhizome. The presence of fruit or the occurrence of heavy winds increases plant losses in the farms. After the pseudostem has fallen a rapid distintegration of the already partially decayed rhizome occurs, leaving an empty pocket in the soil at the site of the former rhizome. This hole at the center of the followers makes it impossible for them to support themselves as adequately as is normally the case. As a result they are particularly susceptible to damage from high winds even if not weakened by decay.

One of the peculiarities of the disease is that the plant can become weakened to such an extent that it is ready to fall over without any pronounced symptoms appearing on the leaves or fruit. It must be inferred from this that much of the vascular tissue continues to function up to the latest stages of the disease and that if the disease is caused by an organism, it is not primarily a vascular invader.

The symptom that actually distinguishes head rot is not, however, the outward appearance of the plant but the presence of decay in the rhizome of a still living plant. In its earliest stages it appears as a watery discoloration of the normally white rhizome tissue. In sectional view, the infected area is frequently outlined by a ring of darker discoloration ranging from dark brown to black with traces of red.

In all mature plants the decay has been found in association with the tunnels of the banana weevil borer, Cosmopolites sordidus (Germ.)[2]. As a result losses in the past have generally been attributed to the infestation of the rhizome by the weevil borer. Only in new plantations from three to eight months old showing no signs of fallen pseudostems is the decay regularly found in the absence of the borer tunnels.

The decay commonly passes from an infected "seed piece" or "mother" rhizome to its follower via the neck joining the two. Infection also appears to occur directly from the soil since in all cases in which the decay cannot be traced to the "mother", the decay in its initial stages can be found only in the cortex in close association with the epidermal layer. In later stages the decay is found in both the cortex and central cylinder though the decay does not pass readily from one to the other.

No fungal mycelium has been found in any of the sections cut from tissue in the early stages of decay. Cultures made from the decayed tissue indicate that bacteria are always present.

An investigation of the cause and means of control of this disease are now under way.

STANDARD FRUIT AND STEAMSHIP COMPANY, RESEARCH DEPARTMENT, LA CEIBA, HONDURAS

[1]Investigation by staff members of the Midwest Research Institute, Kansas City, Missouri for the Standard Fruit and Steamship Company, New Orleans, Louisiana.

[2]Identified by Rose E. Warner, Division of Insect Identification, Bureau of Entomology and Plant Quarantine, Washington, D.C.

CUCUMBER DISEASES IN SUMTER COUNTY, FLORIDA

By John H. Owen and Dixon Connell

Sumter County is one of the oldest cucumber growing areas in Florida with approximately 1000 acres being grown each season. Cucumbers are planted during the last two weeks of January, and frost may occur up until the middle of March. Growers place long V-shaped wooden troughs along the rows of plants, and when frost or cold winds threaten these troughs are inverted over the plant rows. The following disease survey was made April 6, when most of the growers were making their first and second pickings of the season.

A vascular Fusarium wilt (Fusarium sp.) was first reported in this area in 1949, and since that time it has continued to spread, causing severe losses. Most of the growers for the current season have planted cucumbers on new land in attempts to escape the disease. However, some growers moved their wooden troughs from infested land to the new fields, thus apparently introducing the fungus. On new land where wooden troughs were brought from infested soil there was about 1 percent infection scattered throughout the field. Cucumbers planted on infested land for the second and third successive years resulted in 25 to 75 percent infection at the beginning of the picking season. This disease has not been reported from other cucumber growing areas.

Downy mildew, (Pseudoperonospora cubensis (Berk. & Curt.) Rostow.) appeared to be well under control in fields where growers were spraying twice weekly with nabam and zinc sulfate or dusting with zineb. In fields where growers were alternating with zineb and copper dusts or were not following a rigid spray program, downy mildew infection ranged on the average from 10 to 20 percent. Only one field had 100 percent infection with all of the plants being severely infected.

Anthracnose (Colletotrichum lagenarium (Pass.) Ell. & Halst.) seemed to be rather uniformly distributed throughout the entire area, causing more damage than any other disease, and appeared to be much more severe than in previous years. Eighty percent of the fields had from 20 to 30 percent infection. One four-acre field showed 100 percent infection with most of the plants dying before the first picking.

Powdery mildew (Erysiphe cichoracearum DC.) and angular leafspot (Pseudomonas lachrymans (E.F.Sm. & Bryan) Carsner) were not observed in any of the fields.

Wind and Cold Damage: High winds on March 25 caused considerable damage to the tender foliage throughout the entire area, injuring 90 percent of the plants. Cold injury to fruits was common in the colder sections, affecting up to 33 percent of the cucumbers harvested during the first and second pickings.

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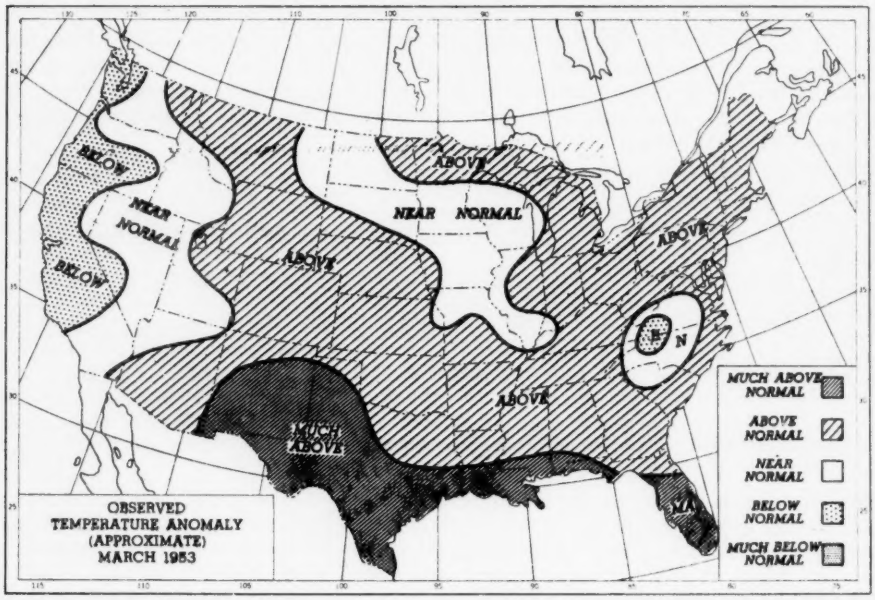

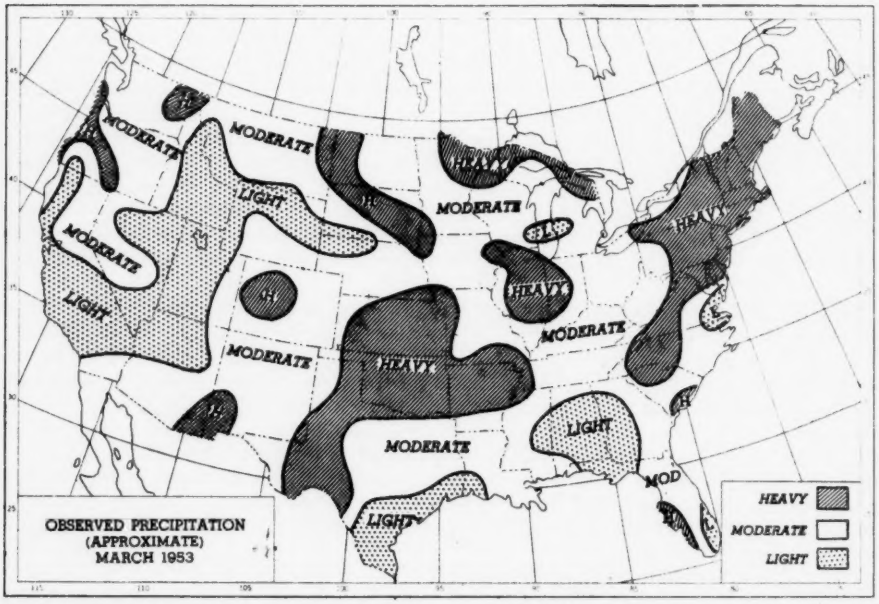

The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.